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THE AMERICAN JOURNAL OF ROENTGENOLOGY

Editor, James T. Case, M.D., Battle Creek, Mich.

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SOME ESSENTIAL POINTS IN THE ANATOMY OF THE LUNG*

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MADISON, WIS.

THE gross distribution of the bronchi within the lungs is familiar to all who have made a study of these organs. I therefore pass over a detailed description of the bronchial tree and proceed to certain points connected with the finer structure of the lungs.

If the bronchial tree be followed to its ultimate divisions a point will be reached where the bronchi are no longer of a regular outline but they have here and there small air spaces (alveoli) opening into their lumen, such bronchi are termed *bronchioli respiratorii*. Each bronchiolus respiratorius divides into branches which have a still greater number of alveoli connected with them, the *ductuli alveolares*. Each ductulus alveolaris is connected with a variable number of irregularly spherical spaces which also possess alveoli, the atria. With each atrium a variable number (2-5) of *sacculi alveolares* are connected which bear on all parts of their circumference alveoli, the *alveoli pulmonum*.

We find then that, beginning with the bronchioli respiratorii, alveoli are connected with all the remaining divisions of the bronchial tree and with the air spaces connected with them.

The last division of the bronchial tree before it breaks up into the parenchyma of the lung is the ductulus alveolaris. We may, therefore, consider it and the air spaces connected with it as representing a special area; moreover, it will be found that the entire lung is built up by the constant repetition of this area which is termed the (primary) lobule of the lung.

The primary lobule of the lung may be defined more exactly as that area within the lung which consists of a ductulus alveolaris, the air spaces connected with it, their blood vessels, lymphatics and nerves (Plate 1).

Some authors, notably those belonging to the French school, apply the term "lobule" to those larger areas, composed of a so-called "intralobular bronchus" with its subdivision into bronchioli, bronchioli respiratorii, ductuli alveolares, the air spaces connected with them, their blood vessels, lymphatics and nerves. In other words these "lobules," composed as they are of groupings of from 50 to 200

* Abstract of an address delivered at the Seventeenth Annual Meeting of the American Roentgen Ray Society, Chicago, Ill., Sept. 27-30, 1916.

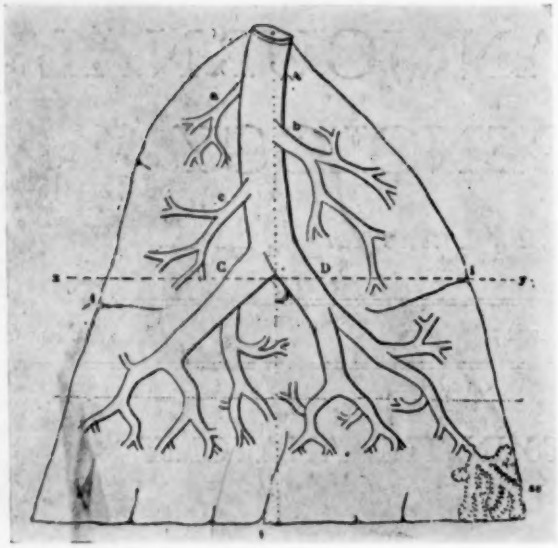

FIG. 1. SCHEMATIC OUTLINE OF A SECONDARY LOBULE, SHOWING AN INTRALOBULAR BRONCHUS AND ITS SUBDIVISIONS.

In the lower right hand corner an attempt has been made to show the ultimate arrangement of the air spaces. (After Laguesse.)

primary lobules, are best described as secondary lobules (Fig. 1).

Beginning with my study, in 1906, of the distribution of the blood vessels to the pleura pulmonalis, I have come to realize that we must distinguish between lungs which have a thin pleura, as in the cat, dog, rabbit, and rat, and those lungs which have a thick pleura, as in the ox, sheep, pig, and man. In the former group the secondary lobules are ill-defined; in fact only an intimate knowledge of lung structure permits of their being recognized. In the latter group the connective tissue septa are well defined so that the boundaries of the secondary lobules are easily recognized (Fig. 2); but with a knowledge of the arrangement of the air spaces the primary lobules are readily made out, especially if the sections be 40 micromillimeters or more in thickness (Fig. 2). The unit of structure in all lungs is the primary lobule.

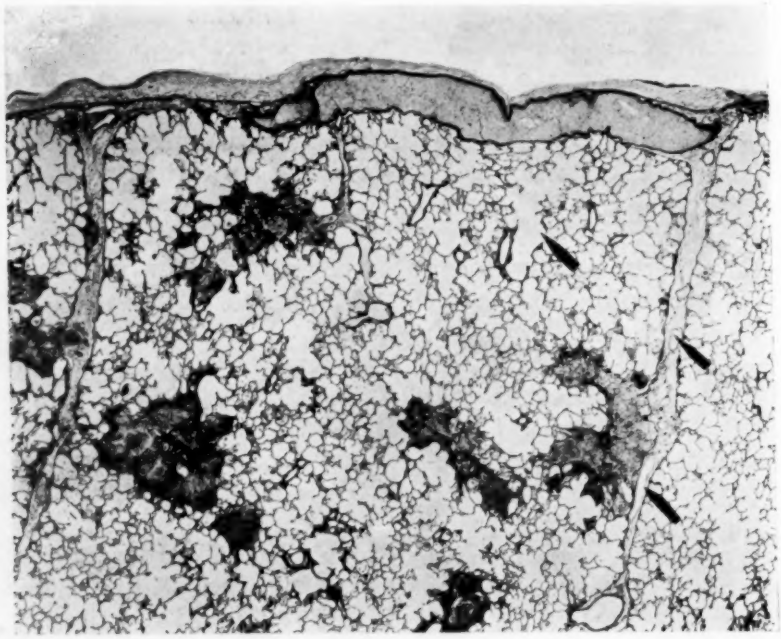

FIG. 2. PHOTOGRAPH OF A SECTION OF THE HUMAN LUNG TAKEN PERPENDICULAR TO THE PLEURA.

The septa mark out a secondary lobule. The two indicators at the right point to lymphatics situated in the septum. The single indicator within the secondary lobule points to a primary lobule. Portions of other primary lobules can be easily made out. Note the well distended lymphatic in the pleura.

This arrangement of pulmonary tissue occasions under certain pathological conditions areas of increased density on *x*-ray plates which have a more or less triangular outline, when viewed from the side. Take for example a case of tuberculosis in which the lung has been removed, inflated and photographed. In a flat picture the irregular triangles ("fans") can be made out, where the disease has not progressed too far, not only at the periphery of the lung but also in the deeper portion of the lung. If in place of a flat plate we use stereoscopic plates, the picture is all the more pronounced. The size of the triangle depends on whether one or more primary lobules are involved, or whether one or more secondary lobules are involved. The type of tuberculosis also influences the character of the density. In advanced stages the picture is one of diffused density and often a lung will show the irregularly shaped fans in one portion and the more advanced diffused density in another portion.

In Fig. 3, lines have been drawn about various subdivisions of the bronchial tree to indicate why the areas of density take a triangular shape.

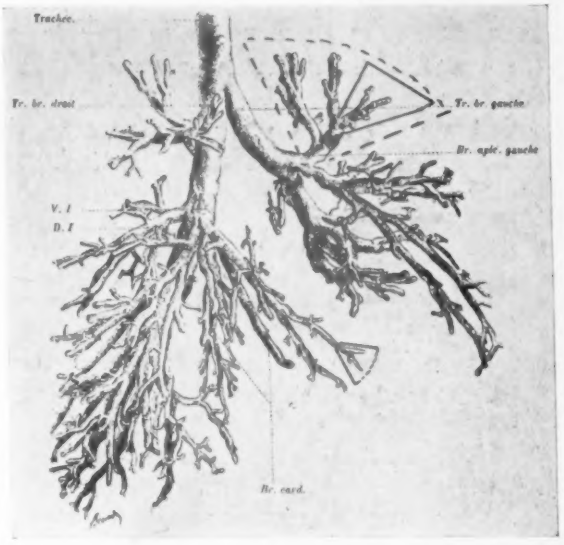

FIG. 3. BRONCHIAL TREE WITH LINES DRAWN ABOUT VARIOUS BRONCHI TO SHOW THAT THE NATURAL EXPLANATION OF THE TRIANGULAR AREAS OF DENSITY SEEN ON X-RAY PLATES IS THE ARRANGEMENT OF THE AIR SPACES.

BLOOD VESSELS

There are two sets of blood vessels to be considered in connection with the lung: the bronchial and the pulmonary. The former are the nutrient vessels of the lung; the latter are the functional vessels.

Bronchial Artery.—The bronchial artery is distributed to the walls of the bronchi, the connective and the lymphoid tissue of the lung. It also supplies the lymphoglandulæ of the hilum. In those lungs which possess a thick pleura and have the secondary lobules marked off by pronounced septa as in the lung of the ox, the sheep, and man, the bronchial artery extends to the pleura and there furnishes a special blood supply to the walls of the lymphatics. In those lungs which have a thin pleura as the lung of the cat, the dog, and the rabbit, it is the pulmonary artery which supplies the pleura with blood.

Bronchial Veins.—True bronchial veins are found only at the hilum of the lung. They empty into the vena azygos, the vena hemiazygos, or one of the venæ intercostales. In all other situations the blood brought to the lung by the bronchial artery is returned by the pulmonary veins. There are no anastomoses between the bronchial artery and the pulmonary artery. The relation of the bronchial artery to the pulmonary vein will be taken up when describing the origin of the pulmonary vein.

Pulmonary Artery.—The pulmonary artery follows in all of its subdivisions the subdivisions of the bronchial tree. As it arches over the main stem bronchus, it comes to lie posterior (dorsal) and slightly lateral to the bronchus. Owing to this position the lateral wall of the bronchus not infrequently shows an increase of density over the mesial wall on roentgen ray plates. At its terminal ending the artery occupies a central position within the lobule (Plate 1, *art.* and Fig. 4), dividing into as many branches as there are atria

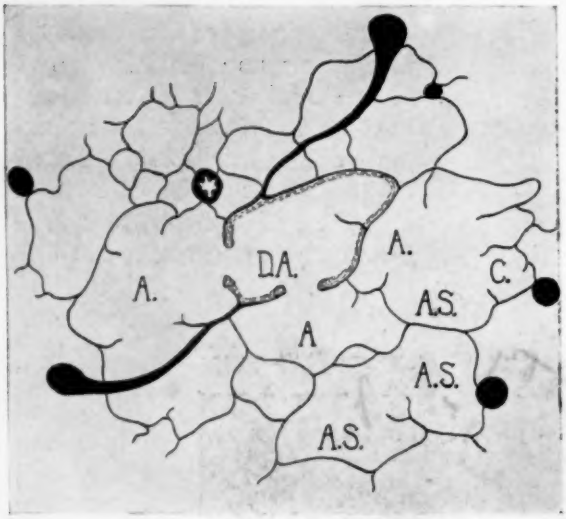

FIG. 4. COMBINATION OF THREE SECTIONS TAKEN THROUGH THE CENTER OF A PRIMARY LOBULE OF THE LUNG OF A DOG.

D.A., Ductulus alveolaris; *A.A.A.*, Atria; *A.S.*, *A.S.*, *A.S.*, Sacculi alveolares; *C.*, Alveolus pulmonis. The tunica muscularis of the ductulus alveolaris is indicated by the broken lines. The pulmonary artery is indicated by the vessel with a stellate opening; the pulmonary veins are in solid black. Note the two veins which arise from the ductulus alveolaris; they correspond to (*2*), in Plate 1. Camera lucida drawing.

connected with the ductulus alveolaris belonging to that particular lobule. Each atrial artery divides into branches which pass to the sacculi alveolares in the walls of which they break up into the capillary network of the lung.

Pulmonary Vein.—The pulmonary veins arise from radicles situated in the pleura (Plate 1; *1*), from the distal end of the ductuli alveolares (Plate 1; *2*), from the place where bronchi divide (Plate 1; *3*) and from the network of capillaries into which the pulmonary artery breaks up. While the pulmonary artery, as already stated, occupies a central position in its relation to the lobule, the veins are situated on the periphery of the lobule (Fig. 4). There is a single exception to this rule, namely: those veins which arise from the distal end of a ductulus alveolaris (Plate 1; *2*) are situated within the lobule (Fig. 4). The blood brought to the bronchi of the lung is returned by the veins which have their origin at the place where the bronchi divide (Plate 1; *3*) and from the distal end of the ductuli alveolares (Plate 1; *2*).

We have seen that the pulmonary artery and its subdivisions follow closely the subdivisions of the bronchial tree lying in close apposition to the bronchi. This is not the case with the subdivisions of the pulmonary vein; they are always situated as far from the bronchi as possible. I wish to lay emphasis on "as possible," for in some situations they approach close to the bronchi but never, in the distended lung, as close as the pulmonary artery.

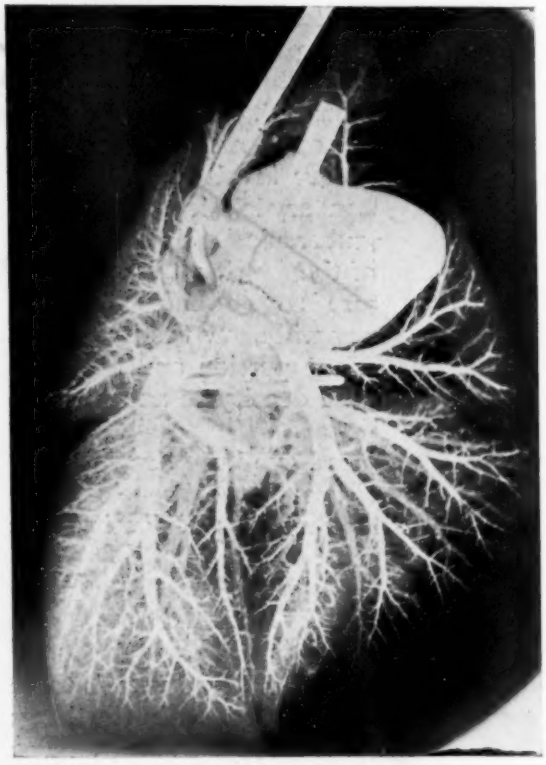

FIG. 5. ROENTGENOGRAM OF THE LUNG OF A DOG IN WHICH THE PULMONARY ARTERY AND THE PULMONARY VEIN HAVE BEEN INJECTED.

By the use of injection masses which give different grades of density the two systems are easily differentiated. The denser shadow is the artery, the lighter one the vein. As the artery follows the bronchi in their subdivisions the relation of artery and vein to the bronchial tree is easily made out. In this and the preceding roentgenogram, the natural curvature of the lung should be taken into consideration when studying either the bronchial tree or the vascular tree.

The relation of the main venous trunk to the bronchus is also different from that of

the arterial trunk; it is situated anterior (ventral) and mesial to the main stem bronchus. Moreover, its course differs in each lung, being more oblique in the right lung than in the left lung (Figs. 5 and 6).

LYMPHATICS

If the preceding description of the blood vessels has been thoroughly understood, the description of the lymphatics will be easily comprehended, for they follow closely the distribution of the blood vessels.

Lymphatics of the Bronchi.—The lymphatics form within the walls of the bronchi a rich network which extends throughout the entire bronchial tree. This network communicates freely with the network of lymphatics which accompany the pulmonary artery; it also gives origin to lymphatics which leave the bronchi at the place where they divide (Plate 1; *3*) and at the distal end of the ductuli alveolares (Plate 1; *2*), accompanying the veins which arise at the same place. Beyond the ductuli alveolares no lymphatics are found; in other words, no lymphatics are present in the walls of the air spaces.

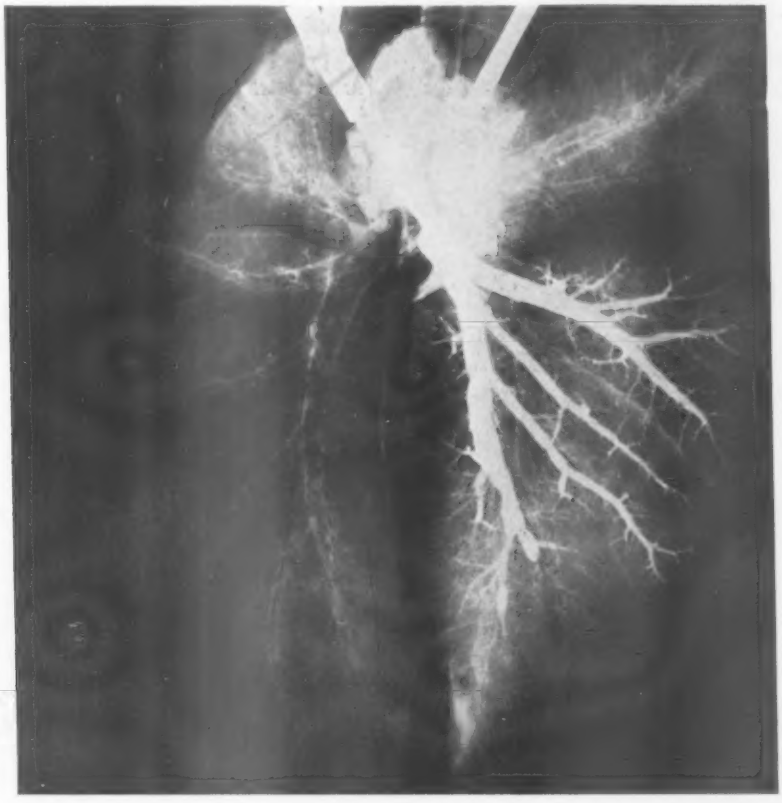

FIG. 6. ROENTGENOGRAM OF A PIG'S LUNG IN WHICH THE PULMONARY VEIN IN THE LOWER LEFT LOBE HAS BEEN PARTIALLY INJECTED.

Note the relation of the vein to the bronchi. Where the injection is complete it can be seen that the vein is spaced nearly equidistant from the adjoining bronchi. On the right side the main stem bronchus with its branches are seen.

Lymphatics of the Pulmonary Artery.—The larger branches of the pulmonary artery are accompanied by two or three main lymphatics, which are so arranged that one of them lies between the artery and the bronchus. These main trunks are connected by numerous loops and there is thus formed a network with a long mesh.

The smaller divisions of the artery are, as a rule, accompanied only by a single lymphatic. The bronchial lymphatics and the arterial lymphatics are in communication with each other at the place where bronchi divide and at the distal end of the ductuli alveolares.

Lymphatics of the Pulmonary Veins.—I have just stated that the bronchial lymphatics and the arterial lymphatics communicate with each other at the distal end of the ductuli alveolares and, someplace and eventually join the lymphatic network about one of the main venous trunks (Plate 1; *3*). The lymphatics about the pulmonary veins also communicate with the pleural lymphatics (Plate 1; *1*). While throughout the lung the lymphatics are, as a rule, destitute of valves, a valve is present at the junction of the venous (deep) lymphatics with the pleural (superficial) lymphatics. This valve opens toward the pleura (Fig. 7).

I have previously called attention to

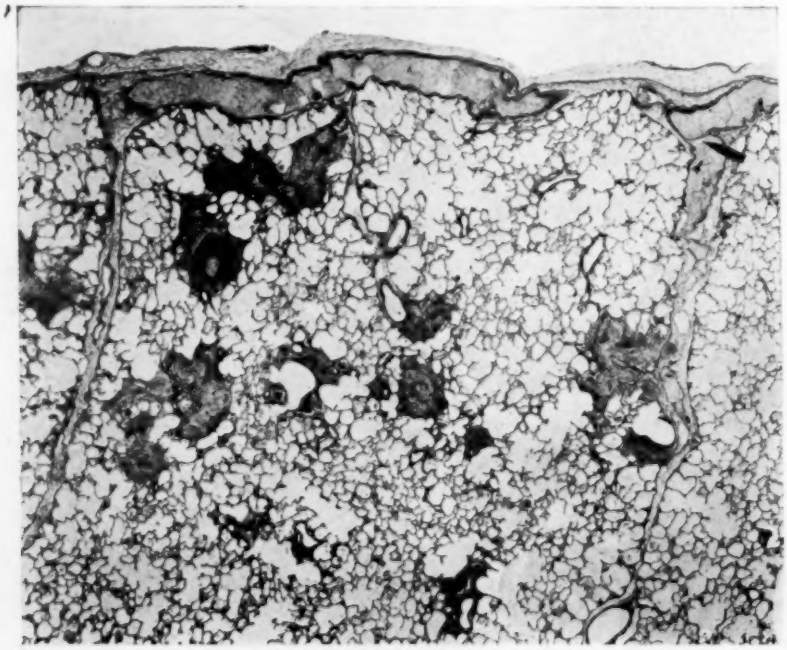

FIG. 7. PHOTOGRAPH OF A SECTION OF A HUMAN LUNG TAKEN PERPENDICULAR TO THE PLEURA.

The indicator points to one of the leaves of the valve situated at the junction of the deep lymphatic with the superficial lymphatic. This valve opens towards the pleura; just below this valve another is seen which also points towards the pleura. The distention of this lymphatic is probably caused by the tubercle which is compressing the septum. The large oval opening at the lower end of the septum is the pulmonary vein.

what earlier, that lymphatics also left the bronchial network at the same place and passed to the two small veins which arise from the ductuli alveolares at this point (Fig. 4). If we follow these lymphatics we find that they join the network of lymphatics about one of the venous trunks on the periphery of the lobule (Plate 1; *2*). The lymphatics that leave the bronchi at the place where they divide follow the veins that arise at the same

the distinction that must be drawn between lungs which have a thick pleura and those which have a thin pleura, and that only in lungs with a thick pleura were the connective tissue septa marking out the secondary lobules well developed. The bronchial artery follows these septa on its way through the lung to the pleura and now we find that the presence of these septa has its influence on the distribution of the lymphatics.

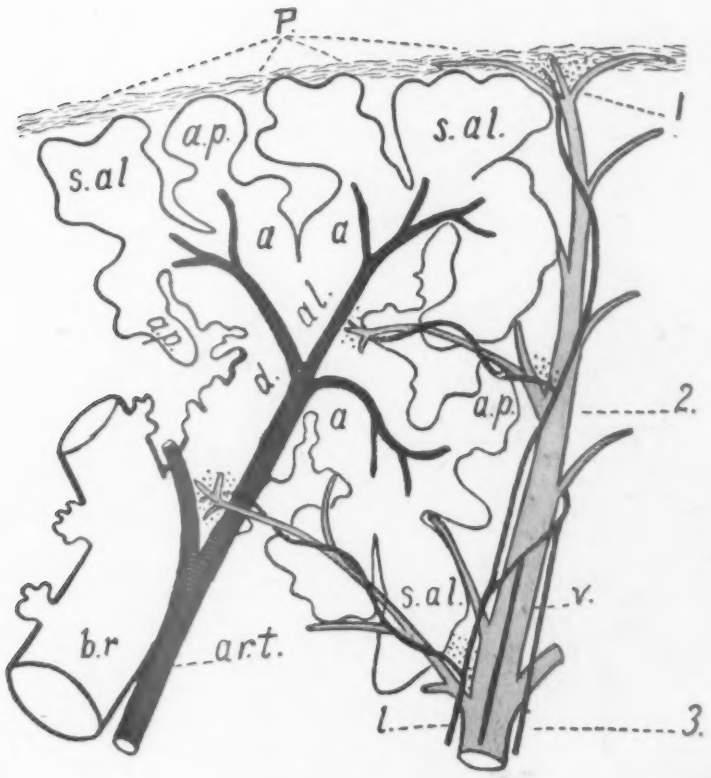

PLATE I. SCHEMATIC LONGITUDINAL SECTION OF A PRIMARY LOBULE OF THE LUNG (ANATOMICAL UNIT), SHOWING THE RELATION OF THE BLOOD VESSELS TO THE AIR SPACES AND TO THE PLEURA, THE POSITION OF LYMPHOID TISSUE AND ITS RELATION TO THE AIR SPACES, BLOOD VESSELS, LYMPHATICS AND PLEURA.

Pulmonary artery, red; pulmonary vein, blue; lymphatics, black. The position of lymphoid tissue stippled in black. The lymphatics of the bronchial system and those of the artery are not indicated. *b.r.*, bronchiolus respiratorius which divides into two ductuli alveolares, *d.al.*, only one of which is carried out in detail; *a.a.a.*, three atria each of which communicate with sacculi alveolares *s.al.*, around the periphery of which are situated the alveoli pulmonum, *a.p.*; *P.* pleura pulmonalis.

Councilman has figured and described lymphatics in these septa with valves pointing toward the pleura, but he did not associate them with the venous trunks which arise from the pleura and pass along these septa. At the meeting of the Association of American Anatomists, held at Chicago in January, 1908, I demonstrated the lymphatics of the human lung and showed that the lymphatics in these septa *were* associated with the venous trunks and that there was a rich network of lymphatics extending from these veins into the septa. In sections taken transverse to the secondary lobules, the lymphatics form a network which surrounds the veins on the periphery of the secondary lobules and then extends to the next vein and so on until the entire secondary lobule is enclosed by a network of lymphatics. In other words we find here, only on a much larger scale, the same arrangement of blood vessels and lymphatics that are found in the primary lobules.

In Fig. 2 some of these lymphatics are shown in one of the septa cut in various planes. Just to the left of the center of the photograph one of the septa belonging to a primary lobule may be seen and within it a portion of a lymphatic cut longitudinally.

Lymphatics of the Pleura.—In the pleura there is a very rich network of lymphatics which contain numerous valves. This network communicates with the deep lymphatics of the lung which extend to the pleura along the pulmonary vein, but the presence of valves (Fig. 7) at the point of union permits of lymph flow, or injection masses, in only one direction. It is only in occasional instances that the valves can be forced and injections made to enter the deep lymphatics from the superficial network. Previous statements that I have made on this point have been misconstrued.

To one unaccustomed to the size of lymphatics when distended, the lymphatics of the pleura seem quite large. Although the microphotographs are from

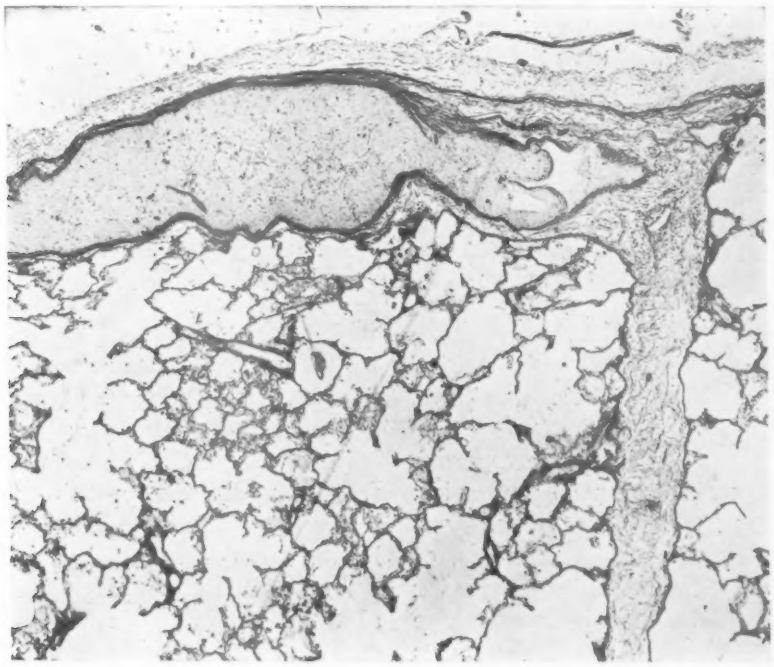

FIG. 8. SECTION OF THE PLEURA OF THE HUMAN LUNG, SHOWING ONE OF THE NUMEROUS VALVES.

Section was stained with Weigert's elastic fiber stain and alum carmine.

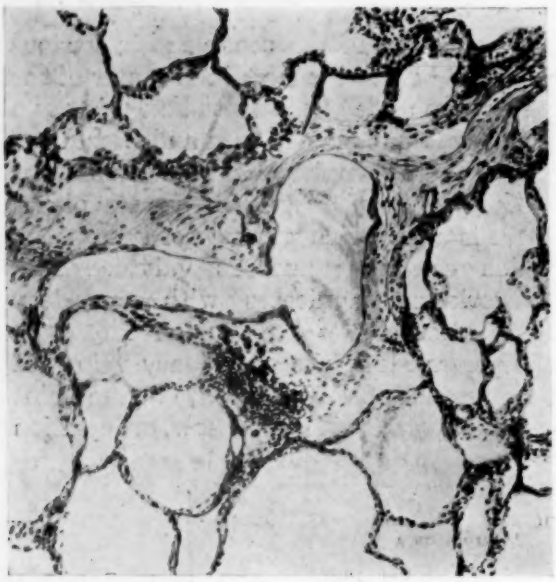

FIG. 9. SECTION OF A HUMAN LUNG, SHOWING THE PRESENCE OF LYMPHOID TISSUE AT THE JUNCTION OF A VEIN COMING FROM THE PLACE WHERE A BRONCHUS HAS DIVIDED, WITH A LARGER PULMONARY VEIN WHICH IS SITUATED IN A CONNECTIVE TISSUE SEPTUM BETWEEN TWO SECONDARY LOBULES.

sections of a tubercular lung, the lymphatics are no larger than I have found them in normal lungs. Fig. 8 shows, more highly magnified than the other two microphotographs, one of the numerous valves present in the pleural lymphatics.

Direction of Lymph Flow.—In the lymphatics of the bronchi, of the arteries, of the main venous trunks and the greater part of the pleura, the flow is toward the hilum of the lung. In the lymphatics about the veins, the flow, in those vessels which are situated just beneath the pleura and communicate with the pleural network of lymphatics, may be toward the pleura. This probably explains why we may find tubercles in the pleura and none in the deeper part of the lung.

In Fig. 7 a portion of a lymphatic in one of the septa is seen well distended. This distention is undoubtedly due to the fact that the normal course of the lymph flow has been interfered with by the growth into the septum of the tubercle on the left of the septum.

Cunningham has recently demonstrated, from the embryological standpoint, that the lymphatics coming from the pleura covering the inferior half of the lower lobe pass through the ligamentum pulmonale and drain into preaortic lymph nodes. I am free to confess that I have missed these lymphatics in the adult, though Willis figured them in his illustration of the pulmonary lymphatics, published in 1675. Their course corresponds to the distribution of the bronchial artery as I have found it in the ligamentum pulmonale of the sheep.

It must not be inferred that any doubt exists in my mind as to the presence of these lymphatics. I have every reason to believe in their presence. Working as I have on the adult lungs, and after their removal from the body, it is quite natural that I failed to find them.

LYMPHOID TISSUE

The distribution of lymphoid tissue within the lung and the relation which it bears to the air passages, the blood vessels, the lymphatics and the pleura should interest not only the pathologist, but also the clinician, for these masses frequently serve as centers to which disease processes may be conveyed through the lymph stream.

Along the larger divisions of the bronchial tree, true lymph nodes have been described and figured by a number of investigators. In each instance they were found in the angle formed by the dividing bronchi. In the normal bronchioli respiratorii and ductuli alveolares I have failed to find lymph nodes or lymph follicles, but have found masses of lymphoid tissues which were situated between the muscle coat and the accompanying branch of the pulmonary artery.

As we have seen in connection with the lymphatics, the place where bronchi divide and the distal end of the ductuli alveolares are important landmarks; so here we find small masses of lymphoid tissue in the

same situation and bearing a direct relation to the lymphatics which arise at these points (Plate 1; *2, 3*). If we follow the veins and their accompanying lymphatics which arise at these points, to their junction with the venous trunks and the accompanying lymphatics, there will be found, in the angle formed by the union of the two veins, lymphoid tissue (Fig. 9). Veins which arise from the capillary network in the walls of the air spaces do not possess lymphatics or lymphoid tissue.

When lymphoid tissue is present along the course of the pulmonary artery it does not, in normal lungs, form the sheathlike arrangement described by some authors, but is found as small masses situated between the artery and air spaces rather than between the artery and bronchus.

In the pleura we always find a small mass of lymphoid tissue associated with the place where the radicles of the pulmonary vein unite to form a venous trunk and the lymphatics associated with the venous trunk join the pleural network of lymphatics (Plate 1; *1*). The amount of lymphoid tissue present at this point varies with age of the individual and the amount of pigment present; being increased in amount the older the individual and the greater the quantity of pigmentation (Fig. 10).

Lymph nodes and lymph follicles have been described as present in the pleura. I can not recognize them as normal structures. They are, in my opinion, always pathological, taking their origin from the presence of irritating substances, like particles of carbon; or as an hyperplasia of already existing lymphoid tissue, as in leucæmia. In the latter case pigmentation is usually absent.

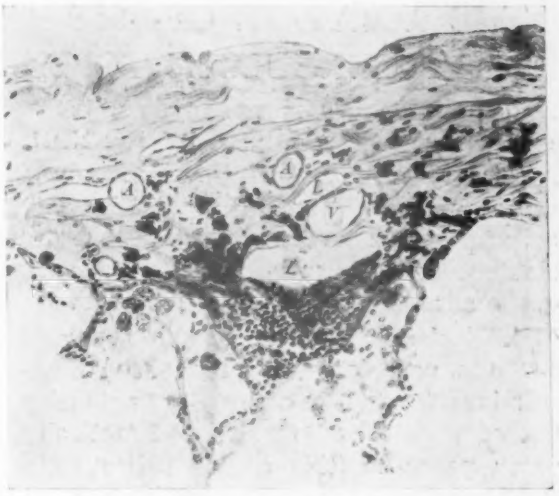

FIG. 10. SECTION TAKEN PERPENDICULAR TO THE PLEURA OF A MAN THIRTY-EIGHT YEARS OLD.

A large amount of pigment is present. *A.A.*, bronchial artery; *V.*, pulmonary vein; *L.L.*, lymphatics. Just below the larger of the lymphatics a mass of lymphoid tissue can be seen.

For the correct interpretation of roentgenograms, definite knowledge of the distribution of the bronchi, the arteries, the veins, the lymphoid tissue, the lymph follicles and lymph nodes within the normal lung and at the hilum is absolutely necessary; for it is impossible to correctly understand the pathological without a previous knowledge of the normal.

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THE RELATION OF THE PATHOLOGY OF PULMONARY TUBERCULOSIS TO THE ROENTGEN FINDINGS*

BY KENNON DUNHAM, M.D.

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CINCINNATI, OHIO

THE claims I make for this study of the pathology of pulmonary tuberculosis are not those of novelty or priority but of review and research. Practically every phase of this question has been touched upon by some one. I did not start to build roads through the wilderness, I started to chart a clear highway from the maze of long existent roads and trails and by-paths. To do this I had to build three bridges; I had to certify three facts based upon verified data. What was required was the judicious interpretation of the characteristic tuberculous markings upon the roentgenogram. I have previously carefully described these characteristic densities, but where they were to be found in the lung and to what pathological lesion they were due was an unsolved problem. To answer these questions it was necessary to know where tuberculosis begins and how it extends through the pulmonary tissue.

Last year I reported that the fan-shaped densities seen upon the roentgen plate were due to masses of tubercles found in the lung near the pleura, and that their shape was due to the anatomical structure of the lung. That is, the triangle was formed by numerous tubercles and took its shape from the anatomy of the portion of the lung involved, not from the shape of the tubercles. To-day I wish to show that the tuberculous lesion starts in the lymphoid tissue within the lung and is spread first through the lymphatics, later by breaking into a bronchus, and later still by penetrating into the veins. Furthermore, while the greater part of the lung is drained by the lymphatics toward the hilum, the peripheral portion of the lung is drained by lymphatics which join those situated in the pleura.

I do not wish to deny that a tuberculous lesion can start in a capillary or another vessel; nor that it may start within some division of the bronchial tree. Lesions have been proved to exist in each of these locations, but I do wish to reassert with emphasis that my research leads me to believe that generally the tubercle bacilli primarily do not cause a lesion in the capillaries or in the air passages, although they must enter the lung by one of these avenues, and that the bacillus is generally removed from the ductulus alveolaris, as is carbon, to the lymphoid tissue centers before the germ has caused a pathological lesion in the air passages.

Obviously a massive or virulent infection within either a vessel or an air-tube, so great that the lymphatic system is overpowered, results in a lesion at such a point. Such lesions occur mostly as the result of experimentation or as late-stage secondary lesions and not as early primary foci. In fact, they are unusual as late secondary lesions.

I said that practically every point of the pathology of pulmonary tuberculosis had been touched upon by some one. As to its beginning in the lung, Kretz confirms Aufrecht in that it begins in the blood-vessels. Birch-Hirschfeld claims that it arises in the mucous membrane of a medium-sized bronchus of the fourth or fifth order; Nichol, in the bronchiolus respiratorius; Abrikosoff holds that customarily tuberculosis

* Read at the Seventeenth Annual Meeting of the American Roentgen Ray Society, Chicago, Ill., Sept. 27-30, 1916.

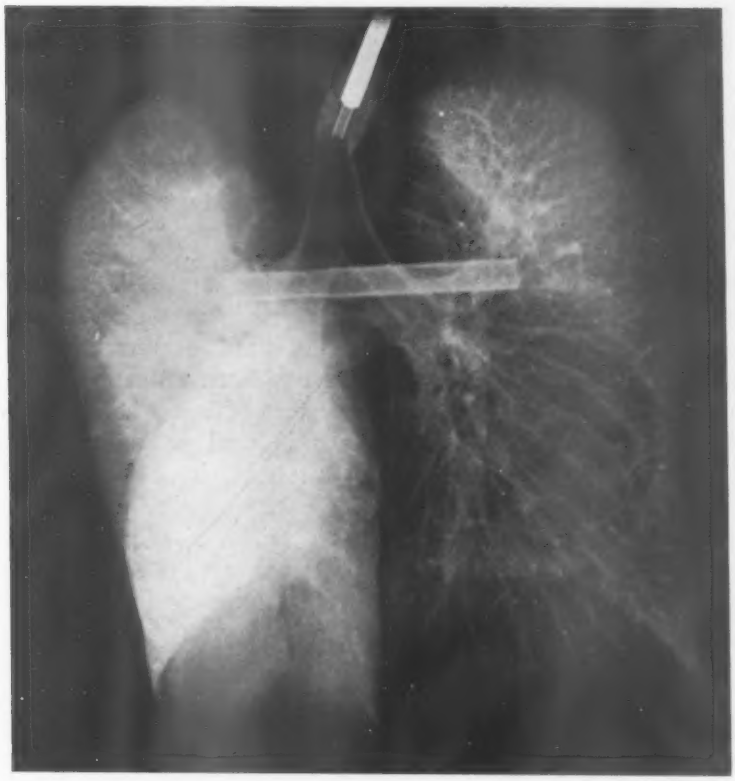

Fig. 1. A print from one of the stereoscopic plates. The left lung is normal; the infarcts are seen on the right side. The circular areas of heavy density seen throughout the left lung at the end of the trunks are not tubercles.

Fig. 2. Photograph of gross lung section showing from where the block has been taken at the edge of the infarct.

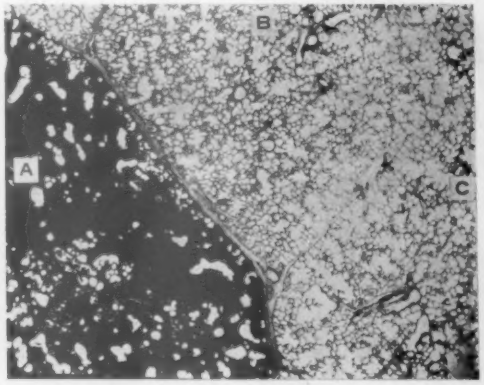

Fig. 3. Reproduction from a microphotograph showing how the trabeculæ wall off the blood. *a*, large amount of blood; *b*, small amount of blood; *c*, no blood.

begins in an interlobular bronchus and the primary lobule lesions arise secondarily by aspiration, while Tendeloo believes they arise peribronchially in the lymph vessels; Virchow, speaking of other organs than the lung, says that the lesion begins as an increase of cells like those of lymphatic glands. Ziegler says it begins peribronchially as an increase of leucocytes and describes these cells as "small round-cells, certainly coming from the blood-vessels." Jordan claims that the disease starts in the bronchial glands at the hilum and extends by retrograde movement through the lymphatics to the parenchyma of the lung. The older clinicians have always claimed in an indefinite way that it started at the apex.

The histological structures which each of these in turn has described are readily noted in my sections and yet it is manifestly impossible that each of these men should be right in his conclusions, differing as widely as they do. However, as I felt that these different investigators were honest and had seen what they considered the truth, I had to go further to find some conclusion that could be honestly drawn from *all* of these previous observations.

Dr. Miller to-day has called your attention to the all-important anatomy of the lungs, without an accurate understanding of which it is impossible to conceive the process of infection, the beginning and dissemination of tuberculosis in the lung. But given this anatomy, you are in a position to follow my interpretation of how these processes take place. I believe that in the vast majority of cases of pulmonary tuberculosis the bacillus enters the air passages with the dust and soot and is carried into the smaller bronchial subdivisions (beyond the ciliated epithelium) as far as the ductulus alveolaris and the ultimate lobule; and that from there it is taken by the large phagocytes or macrophages, before the bacillus has caused any infection at its point of lodgment, and is carried to the lymphoid tissue which is abundant in this locality. If neither the phagocyte nor the lymphoid tissue is able to destroy the invader, or if the lymphatics cannot sufficiently drain the part, a tuberculous lesion develops in or near the lymphoid center which at first is only an abnormal increase of these lymphoid cells. If the bacilli grow instead of being destroyed, it indicates a greater effort on the part of the economy resulting in the appearance of epithelioid and giant cells. Necrosis, caseation and the formation of cavities will follow in their wake.

This is the tubercle, but it does not start, as has been often claimed, in the necrotic centers of Nichol, which are usually pneumonic and secondary areas, but in the lymphoid tissue from which the lymphoid cells extend like pseudopodia through the interstitial tissue, surrounding the neighboring air cells.

In the acute form there is little destruction of the elastic fibers of the alveolar walls, and there is evidence of exudation into the alveoli in proportion to the virulence of the infection. In such cases the alveoli contain cells which have been variously identified as epithelial, endothelial and epithelioid.

Giant cells and necrosis are not a necessary part of the acute stage of the lesion. In the chronic form necrosis has taken place, the elastic tissue fibers are to a large extent destroyed, epithelioid cells and giant cells surround the necrosis and these in turn are surrounded by small round-cells and connective tissue cells.

In either case the lesion can be followed along the lymphatics of the veins, the arteries and the bronchi. These lymphatics are easily made out by their engorgement or by the pigment deposited in the alveolar connective tissue just outside their walls. Thus, the course of the tubercle bacillus from the bronchus to the parenchyma of the lung coincides with that followed by the dust and particles of carbon, but the coal pigment is received without causing grave pathological changes, while the tubercle bacillus lives, grows, multiplies and destroys.

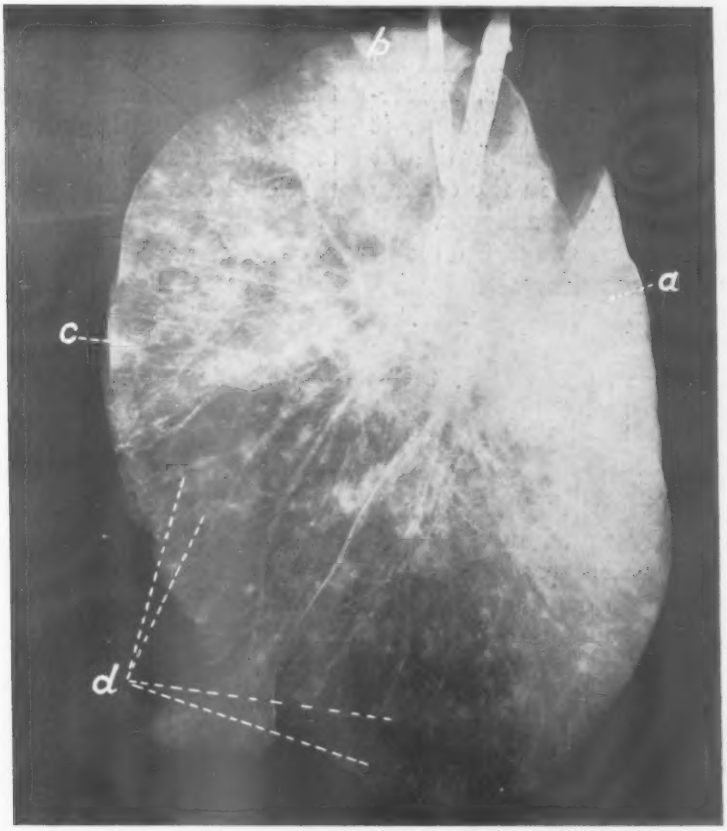

Fig. 1. A print from one of the stereoscopic plates: *a*, with hemorrhage complication in the apex of the lower lobe; *b*, chronic tuberculosis at apex of the upper lobe; *c*, acute tuberculosis from which block was removed showing microscopic abscesses, microphotograph of which is shown on Plate B, Fig. 3; *d*, typical tuberculous fans which stereoscopically can be seen to come to the pleural surface.

Fig. 2. Gross section of lung, showing area from which block was taken and old lesion.

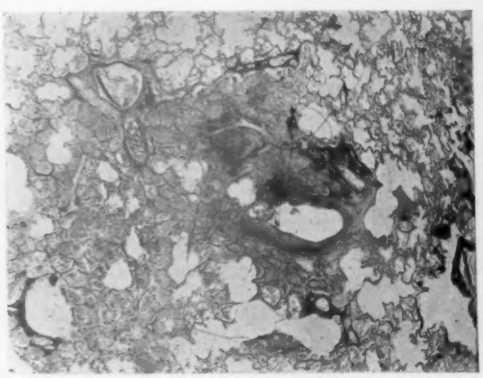

Fig. 3. Microscopic section showing minute cavities. By a study of the serial sections these cavities were found to drain into the bronchus.

Thus we have the invasion, the infection and dissemination, how it gets in, how it takes root, and how it may be disseminated along the lymphatics. Now if necrosis takes place and the node softens, disintegrates and breaks into the bronchus, it may be very easily spread by aspiration at this secondary stage. Late in the disease the tubercle often breaks into a blood vessel and may be spread by means of the blood current. Thus we have infection by inspiration, aspiration and circulation. When the infection takes place by inspiration the apices are most frequently involved.

Let us now examine how this process will explain the various modes of infection previously referred to.

The clinician dealing with the physical signs most frequently found evidence of his lesion at the apex and secured abundant pathological material to confirm his idea.

Jordan examined the chests of children roentgenographically, finding his earliest lesion at the hilum. By his pathological research he easily proved that the glands are much more extensively involved than the parenchyma and that there is a peribronchial inflammation.

Ghon of Prague recognizes pathologically all that Jordan has seen roentgenologically, but conclusively proves that the original lesion was in the parenchyma of the lung, and not in the bronchial gland.

Ziegler recognizes exactly the primary seat of the tuberculous focus, and describes it to be where we know lymphoid tissue exists normally but fails to emphasize the importance of that lymphoid tissue.

Virchow emphasizes the importance of the lymphoid tissue in the intestines and liver, but fails to mention its significance in the lung.

This study emphasizes the fact that lymphoid tissue plays the same part in the lung as in the other parts of the body; that is, lymphoid tissue is always so placed as to form a filter through which lymph from the periphery must pass before it is admitted to the central ducts and the blood stream.

Tendeloo lays great stress upon lymphatic vessels but overlooks the value of the lymphoid tissue.

The work of Virchow, Ziegler, studied in the light of the researches of Tendeloo, is the pathological highway I wish to chart for you to travel.

Nichol carefully describes the anatomy of the lung, but does not differentiate between vein and artery. He only casually mentions the lymphatics and nowhere mentions lymphoid tissue. He describes the lesion as arising in the bronchiolus respiratorius. Had he known of the existence of the lymphoid tissue as an important anatomic structure he could not have failed to recognize from his serial sections the important part it plays in the beginnings of tuberculosis. Abrikosoff was just as far away from this important fact as was Nichol.

Birch-Hirschfeld's illustrations show that the lesion, which he describes as starting in the mucous membrane of the bronchus, lies at a bifurcation of the bronchus and lymphoid tissue, as such a bifurcation is always found just outside the bronchus. Furthermore, one of Birch-Hirschfeld's illustrations shows a fan-shaped induration extending from the lesion at the bifurcation to the periphery.

Aufrecht and Kretz believed that the lesion starts in the blood vessels. Aufrecht studied the larger veins by histological sections and Kretz by the injection of tubercle bacilli into the circulation of animals. If Aufrecht had studied serial sections, he would have seen that the lesion extended from the lymphoid tissue outside of the vessel into the lumen and did not start from the endothelial cells. Kretz injected such large numbers of bacilli as to cause emboli in the capillaries and only rarely could such a process take place in the human economy.

These various opinions resemble those of the blind men who described the elephant as like a tree-trunk, a rope, or serpent, a spear, etc., according to what they sensed from their personal contact with

PLATE C. ADVANCED DISSEMINATED TUBERCULOSIS INVOLVING ALL LOBES.

Fig. 1. A print from one of the stereoscopic plates. *a*, area from which block was taken out of lower right lobe.

Fig. 2.

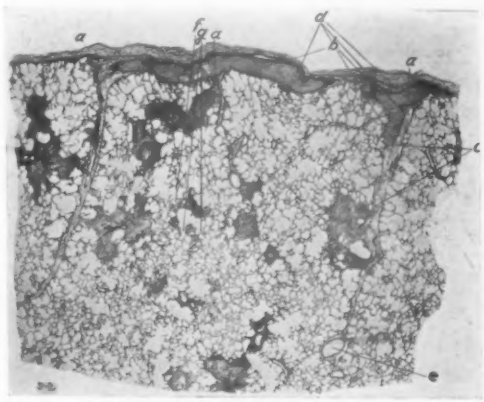

Fig. 3.

Fig. 2. Gross section of lung showing from where block was taken.

Fig. 3. Microscopic section. The lymphatics are engorged and the trabeculæ (*a*) have been able to limit the extension of the tuberculous masses. Active and chronic processes are closely associated in this lung. The lymphatics (*b*) in the pleura are greatly engorged as well as those (*c*) passing toward the pleura with the trabeculæ. Lymphatic valves are seen at (*d*). By a study of the serial sections this lymphatic can be traced from the vein (*e*) to the pleura. This large lymphatic vessel, as well as numerous smaller lymphatic vessels which it drains contains a great number of epithelioid cells, just as similar studies of carcinoma of the lung show the lymphatics filled with carcinoma cells. The numerous valves in the course of this lymphatic prove that the lymph-stream flows toward the pleura. At (*f*) a bronchiole divides. (*g*) represents bronchiolus respiratorius. A study of the serial sections of this area shows that the tuberculous mass surrounding the bronchiole at no place obliterates any part of any of the lobules beyond this bronchiole.

parts of it. All are right, but none discovered the universal truth which is here, the fact of the importance of the lymphoid tissue of the lung.

The lymphoid tissue is situated in the peripheral portion of the lung and also in the pleura. From this outer zone, passing toward the hilum, we have the pulmonary lymph nodes and at the hilum we have the various bronchial groups. Thus, we have three zones of lymphoid structures and these can be easily made out in the roentgenogram. The outer zone is usually the seat of the initial lesion and the most generally involved. Also there is a progressive increase of frequency from the base to the apex, but this must not be construed to mean that it is the only seat of pulmonary tuberculosis. It is unnecessary to go further into the known pathology and classifications of tuberculosis in this paper. Forty years ago Ziegler wrote all that I could say with the three exceptions I spoke of. He did not tell us the location and the importance of the lymphoid tissue as the primary seat of tuberculosis; he did not realize the drainage of these areas by the lymphatics through the trabeculæ to the pleura, and he did not see that the tubercles arrange themselves in the lung so as to form a cone-shaped mass with its base to the pleura and its apex toward the hilum. These three facts or bridges, namely the knowledge of where tuberculosis begins, how it extends through the pulmonary tissue and how the cone-shaped masses are formed, connect our recorded observations, and give us a clear road to our goal.

Arrived here, we easily progress to the interpretation of the characteristic tuberculous markings upon the roentgenogram.

The solution of this entire problem was found not in a pathological, but in an anatomical laboratory. It is to the anatomical laboratory that one must go for fundamentals. This is as necessary for the study of the lungs as roentgenologists have found it for the study of the bones. I am indebted to Dr. Miller of the University of Wisconsin for his instruction and untiring interest in my problem.

But to return to the pathology of pulmonary tuberculosis, another important view of the infection of tuberculosis is that of Dr. Baldwin, whose writings undoubtedly suggest that tuberculous infection does not occur in the adult lung unless there has been a previous infection in childhood. Manifestly neither such a premise nor the converse is subject to proof, but can be reached only by deduction from experimental research. It is known that subcutaneous injections of the bacillus may cause only a slight lesion at the point of inoculation, which in a short time apparently completely heals. But the glands draining that part may undergo a violent reaction which may extend even to the point of suppuration and discharge. A subsequent inoculation into another part of the body of this animal will frequently cause a reaction at the point of the first inoculation much more severe than that caused by the initial dose.

Ghon, as stated before, has undoubtedly shown that in children the primary tuberculous focus in the lung may be histologically very slight and that the glandular reaction at the hilum may be violent, even going to the point of suppuration and discharge. Given now a secondary infection in such a child, although the focus may be in an entirely different part of the lung, it is easy to conceive that a similar process to that which takes place in the twice infected animal might take place in the child. Such a phenomenon might not be completed for years or until adult life. Further it is not necessary that the original inoculation should take place in childhood, but frequent inoculations are necessary for the development of pulmonary tuberculosis. To me this thought embodies the value of Dr. Baldwin's theory and emphasizes the importance of the study of latent tuberculosis.

The last point bearing upon the relation of the pathology of pulmonary tuberculosis to the roentgen plate is the classifica-

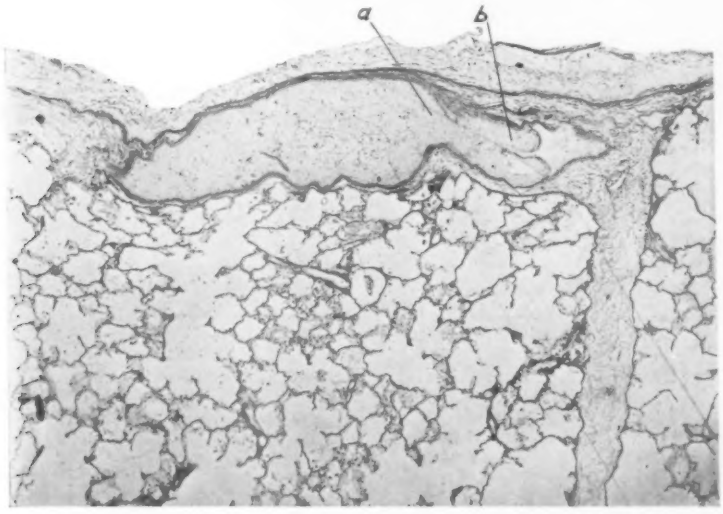

PLATE D. A LARGER MAGNIFICATION OF ANOTHER SECTION FROM THE SAME BLOCK AS PLATE C, FIG. 3.

This plate shows most diagrammatically a large lymphatic valve straining under the back pressure of an engorged lymphatic in the pleura. Miller and Cunningham have previously proved that normally there is a lymphatic flow toward the pleura, and Councilman considers that the second set of lymphatics "play an important part in the pathology of the lung, for it is undoubtedly by this route that infections in the center of the lung extend to the pleura."

tion of tuberculosis. Not that there is any paucity of classifications, but those we have are based on etiology, mode of infection, morbid histology, pathological lesions, localization, physical findings and symptomatology.

What is needed for our purposes is a classification based upon roentgenological findings, and a complete understanding of pulmonary tuberculosis.

EXPLANATION OF ROENTGENOGRAMS

The purpose of these roentgenograms is to illustrate the method by which this pathological research has been prosecuted rather than to prove the involvement of the lymphoid tissue.

The process employed has been to gently inflate the lungs with air and to secure a stereoscopic roentgenogram of each lung. For this purpose only such lungs or lobes of the lung can be utilized as have been removed from the thorax without tearing the pleura. The lungs are then filled with alcohol and the bronchus or trachea tied off and weighted, so that they are completely submerged in 95% alcohol in jars sufficiently large not to compress the lung. Thus the lung is hardened in the same shape and degree of expansion in which the roentgenogram was taken.

Having secured this material the roentgenograms are carefully studied for abnormal densities and their location determined by means of the stereoroentgenograms. It is remarkable with what accuracy these lesions can be found in the hardened lung.

The block is then removed, embedded in celloidin, cut in serial sections, mounted, and studied.

The material has been collected and the roentgenological studies made in the Cincinnati Tuberculosis Sanatorium. I am indebted to Professor Wooley, Pathologist to the Cincinnati General Hospital, for much valuable material. The selection of the blocks, the preparation of the sections and the study of the series have been prosecuted in the Anatomical Department of

the University of Wisconsin under the direction of Dr. Wm. Snow Miller, Professor of Anatomy. Dr. C. H. Bunting, M.D., Professor of Pathology in the University of Wisconsin has given us much valuable advice.

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DISCUSSION ON PAPERS OF DRS. VAN ZWALUWENBURG, MOORE, STEWART, MILLER AND DUNHAM*

DR. FRANK S. BISSELL, Minneapolis, Minn.—Dr. Moore's paper is a model of succinct, scientific statement. I would like to emphazise the importance of the roentgen study of bronchiectasis from a surgical standpoint. Naturally the best surgical results can only be expected when the disease is limited to one of the lower lobes, and this limitation can be determined roentgenologically.

I feel quite incompetent to discuss the papers of Drs. Dunham and Miller, but I know that we owe a debt of gratitude to these men for their efforts to establish the roentgen diagnosis of pulmonary tuberculosis upon a scientific foundation. I am sorry that Dr. Dunham has not more clearly correlated his roentgen signs with the anatomical facts demonstrated by Dr. Miller. I expected him to show that his fan-shaped shadow, or the Sluka triangle, which I take to be the same thing, is caused by an engorgement of the lymphatics within a given lobule. Perhaps he will touch upon this point in closing. I was not aware that the lymphatic structure of the lung had been so neglected by the pathologist and anatomist, but I have had a feeling that not enough attention had been paid to the capillary circulation of the lung. Two years ago, in trying to explain the "net-work" frequently observed in the periphery of the tuberculous lung, I advanced the theory that it was produced by these capillaries, visualized by reason of thrombosis which occurred in the immediate vicinity of tubercles grouped at various points of bifurcation. The point must be emphasized that there is still work to do before the pathognomonic character of various shadows in the tuberculous lung is proven. If we search the lung fields for the "fan" or "triangle" as the one pathognomonic evidence of tuberculosis, we are going to miss many actual cases of this disease. When these signs are present, they undoubtedly mean tuberculosis, but a negative diagnosis should not be made in their absence. If, as Dr. Dunham has suggested, the fan is an evidence of lymphatic engorgement within a given area of the lung it is reasonable to consider it a pathognomonic sign of active tuberculosis.

Dr. Stewart's paper deserves a great deal of discussion and it is only because of the fact that there are so many papers to discuss that little can be said. I have had

* The articles of Drs. Van Zwaluwenburg, Moore and Stewart appeared in previous issues.

three or four cases of pneumonia in children with no physical signs, in which the diagnosis was made roentgenologically. Just why physical signs are sometimes absent in these cases is not clear to me, but since the patients were examined by very competent clinicians I am sure it is not an error to say that there were no physical signs. The evidence in the roentgenograms was very characteristic, and the cases proceeded to resolution by crisis.

Dr. W. F. Manges, Philadelphia, Pa.—The first three papers are deserving of special mention, because they are on subjects not much talked about. Dr. Van Zwaluwenburg's subject is entirely new, and I cannot discuss it. Dr. Moore's paper was complete. It was a splendid presentation and he deserves to be congratulated. Dr Stewart's paper was also excellent. He told us so much that it was impossible to digest it all, but I am sure that when the paper is published, every one of us will read it with much pleasure and profit.

Dr. Miller's work is magnificent. Dr. Dunham's work is a combination of all his former work, and it impresses me that he is going more to the anatomic study than to the true practical tuberculosis study. He would have us believe that the characteristic roentgen appearance of tuberculosis of the lung is a fan-shaped shadow. He tells us that numerous authors disagree as to the origin of the tuberculous infection, and where you find a number of scientific men disagreeing, you can almost put it down as a fact that they are right and that there is no one of them wrong. Certainly, in children, tuberculosis is more prevalent at the hilus than at the periphery. Dissemination takes place by continuity and contiguity, by the blood-stream as well as the lymph-stream. The tubercles break from one lobule into the other. There are well-developed cases of pulmonary tuberculosis in which it is impossible to demonstrate fan-shaped shadows. The physical signs and history are often insufficient to make a diagnosis of tuberculosis. I have a case in mind in which repeated physical examinations failed to disclose tuberculosis. The man finally expectorated blood, and on examining the sputum, bacilli were found. I made stereoscopic plates of his chest. In the upper portion of the right lobe one could almost count the small, calcareous, dense bodies. They were definitely healed. There was no question about it. In the same lobe lower down was a nodular distribution—not fan-shaped, so many nodules that they could not be counted. There was not a great deal of exudate; just these many tubercles. Another patient had a most typical history of pulmonary tuberculosis seven years ago. The right shoulder had been amputated many years before for sarcoma, so that her chest offered unusual opportunities for physical study. She was told that she had pulmonary tuberculosis involving the entire upper right lobe. She took a course of treatments extending over a year and recovered. Since then she has been teaching school and to-day she is apparently as well as any one could be. During the last winter I made some roentgen plates of her chest but found no scar tissue in the upper right lobe.

Now, does tuberculosis exist in the parenchyma of the lung and disappear by dissolution, as does lobar pneumonia, and leave no trace? I am told by pathologists that this is not the case. That once the tuberculosis process is established, it may heal and become quiet, but the evidence never disappears. One bronchus, that going into the right apex, was definitely fixed and almost parallel to it was an area of fibrous tissue with absolutely no infiltration. I could cite case after case of atypical pulmonary tuberculosis just like these. Fan-shaped shadows, whether small or large, can be found in any bronchopneumonic process. You see them in nearly every one of Dr. Stewart's cases. That, it seems to me, is just as applicable for one pneumonic process as

another. The characteristic lesion is the tubercle. It exists no matter where the disease is situated. It is that lesion on which the pathologist bases his report, when he has made a post-mortem examination of a tubercular lung. I do not believe that we can say from a photomicrogram of a tubercle that it will or will not show on the photographic plate. I do believe that we can, from sections shown us to-day, also roentgenograms, conclude that the tubercles, when established, will cast recognizable shadows. I believe that we should depend upon recognizable tubercle shadows in the roentgenographic diagnosis of pulmonary tuberculosis. I do not care what the stage of the disease, except the very, very early stage, or when exudation is excessive, you can always recognize the tubercle shadows, and that shadow is essential to the diagnosis of pulmonary tuberculosis. It does not matter whether the tubercles are discrete or confluent; whether they involve a part or the whole of a lobe.

Dr. George C. Johnston, Pittsburgh, Pa.—In the diagnosis of empyema, I want to warn you against making an error in a case in which the patient has aspirated a foreign body which completely occludes the bronchus. If it has been in there for some time, you need not be surprised if you find the obstructed lung filled with pus. There is no evidence of any lung structure, but the roentgen appearance is that of empyema. Pass a bronchoscope and remove several ounces of pus from that lung, and you will find that is was not an empyema, but a case of obstruction of the outlet.

Dr. L. T. LeWald, New York City.—I want to call attention to two cases, involving the differential diagnosis between appendicitis and acute lobar pneumonia in young children, which I reported some time ago. I have a third case now. In the first two cases the roentgen evidence proved conclusively that it was a pneumonia and not an appendicitis. Therefore, the children were not operated on. In the third case there was a double evidence in this way: the child gave no evidence of exudation in the lung on the plates, and we were fortunate enough to get a shadow in the appendix, showing the presence of a concretion. The child was immediately operated upon. The concretion found in the appendix was roentgenographed after removal and corresponded in shape and density with the shadow noted before operation.

Dr. P. M. Hickey, Detroit, Mich.—The tubercle bacillus does two things. It causes the formation of tubercles, or it may cause an exudative inflammation which may fill up the air vesicles. The roentgen appearances of these two types of tuberculosis may vary considerably.

Dr. William H. Stewart, New York City.—It is not my intention to have any one present interpret my paper as casting reflections on the ability of the clinician in physical diagnosis. I am associated with most capable men who have examined all of these patients carefully and thoroughly and know that when they say physical signs are not in evidence, it is so.

I have never come in contact with a case such as Dr. Johnston describes, but can appreciate that such a condition would give roentgen findings similar to fluid in the pleural cavity.

Dr. LeWald's mention of the symptoms of appendicitis in pneumonia in children again brings out the importance of the roentgen examination of all children suffering from a possible disease within the chest.

Dr. Kennon Dunham, Cincinnati,O.—The practical roentgen value of what I have given you, in my judgment, has been proved correct. If you will take stereoscopic plates, and remember that the time exposure, upon which I have always laid

great emphasis, makes a great deal of difference, you will save yourselves many mistakes and you will get the same results that I have had. If you expose more than two seconds, you will not get good results. You must try to see tubercles before they are too old; it is too late then to make a diagnosis. Nor will you make the mistake of calling a lot of shadows tubercles and have the clinicians laugh at you. If you will take the work I have given you and study it carefully day after day as I do, read your plates well, you will be much more impressed with the value of your roentgen chest work. It is not a question of the value of that reading, but of using it. Practically, it works. I have tried today to give you the pathology of why it works. That the densities are due to tubercles is right; that the shadows are due to tubercles, I agree; but I do resent strongly confusing pathologic and anatomic terms and reading them into the roentgenograms without sufficient basis for proof. You have no right to call a shadow a tubercle shadow unless you know absolutely that that is what it is. I have spent a great deal of time on this work and I am simply giving you what I have found. You can take it for what it is worth. If I have contributed anything at all to our roentgen knowledge of the pathology of tuberculosis, I will feel well repaid for the time and effort. I am convinced that something will come of this sooner or later. In the meantime, I shall go on with my work and report my findings to you from time to time.

DISCUSSION ON PAPER OF DR. MILLS

The paper by Dr. Mills published in the April issue was not accompanied by the discussion. Dr. L. T. Le Wald of New York in discussing Dr. Mills' paper spoke as follows:

"I want to compliment Dr. Mills on his contribution. It was very interesting and forms a firm foundation at last on which we can construct our methods for the diagnosis and treatment of ptosis, but we should not be deterred from looking for pathologic variations and having them properly treated. While I was a surgeon in the Army, I had the opportunity of examining about 72,000 eyes for refractive error. The oculist has the same difficulty in drawing conclusions as to what is normal vision. He must make an arbitrary rule. From the shape of a man's head one may draw some conclusions. From the shape of the head of the President of our society I would say that he may have a myopia, and that our retiring editor has a hypermetropia. What are you going to do? That is the person's build. Will you let it alone? No, you will prescribe appropriate glasses for each case if headache, etc., are present. Another man may have the same or even a greater error of refraction than one of these and yet have no headache and therefore not require glasses. The same thing is true of ptosis. One individual may have a greater degree of ptosis without symptoms than another has with symptoms. We must be guided by these facts in each individual case. If the stomach empties itself within a reasonable time and gives no symptoms, we are not going to subject the patient to a suspension operation even if the greater curvature be in the true pelvis. On the other hand, if the greater curvature be only a few inches below the level of the crest of the iliac bone, and the patient has symptoms referable to a stomach residue and the latter is present on repeated roentgen examination, a suspension operation or a gastroenterostomy may be the only means of curing the patient. A third patient may be perfectly comfortable with a belt, just as a person who wears prisms to correct a strabismus, or a person with a hernia is content with a truss."

PNEUMONOCONIOSIS*

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SAN FRANCISCO, CAL.

PNEUMONOCONIOSIS is the general term applied to the changes occurring in the lungs as a result of the introduction of dust of various sorts into the pulmonary tissue. Three principal groups are recognized—anthracosis, chalicosis and siderosis—according as the dust is derived from coal, stone or iron. A fourth group might include the mixed types of dust to which the city dweller is more or less exposed. As the changes in the lungs are more dependent upon the quantity of dust present than upon the quality, we will not concern ourselves with the individual groups.

Ordinarily, dust inhaled is removed in the upper respiratory tract, but if the amount be excessive, some finds its way into the alveoli of the lungs. Here it is taken up by endothelial phagocytes which attempt to transport the dust particles through the lymph spaces and channels to the peribronchial lymph nodes. In transit, however, "many of the pigment cells appear to be caught in the lymph spaces causing a backing up of similar cells in the spaces behind them. Here the cells seem to act as irritants and cause proliferation of connective tissue," the resulting fibrosis varying in degree according to the amount of dust inhaled and the duration of the exposure.

In early or mild cases, there is merely a diffuse thickening along the course of the pulmonary lymphatics with scattered nodules about the larger accumulations of pigment. In the more advanced cases the fibrosis may become so extensive that dense, airless areas are found scattered throughout the pulmonary tissue. Cavities may occasionally result without tubercular infection.

Clinically, those suffering from chronic dust inhalation are apt to complain first of shortness of breath, asthmatic attacks or chronic bronchitis. As the process advances, the addition of weakness, loss of weight, increasing cough with more or less expectoration, rapid pulse and the finding of impaired pulmonary resonance with râles of various sorts, presents a picture closely simulating pulmonary tuberculosis. The disease in this stage is apt to be progressive, death occurring either from cardiac failure or from a complicating infection.

Roentgenographically, we should expect to be able to detect the pulmonary fibrosis resulting from the long continued inhalation of dust and we are all familiar with the diffuse and uniform increase in the density of the pulmonary markings occurring with advancing years especially in city dwellers. However, I believe that many of us are not familiar with the roentgenographic appearance of the more advanced lesions of pneumonoconiosis and have been reporting these as pulmonary tuberculosis.

The literature seems to be remarkably free from any discussion of this subject. In the standard works on clinical medicine there is no reference to the roentgenographic appearance of the lungs in pneumonoconiosis except in Barker's article in Monographic Medicine in which he states that, "The roentgenogram shows a coarsely granular, evenly distributed mottling of the upper parts of the lung areas, the two lungs being equally affected. The appearance is not unlike that of miliary tuberculosis but the markings are coarser."

The text-books on roentgenography con-

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† From the Department of Roentgenology.

sulted are all silent except Robert Knox and Rieder.

Knox states that "the pigment granules are deposited along the course of the lymphatics, forming nodules and tending by chronic irritation to a marked degree of fibrosis. The lymph glands are enlarged and indurated. In all these types the distinguishing feature in contradistinction to malignant disease is the more uniform distribution of patches of induration all over the lung and bronchial tract."

H. Rieder states that "the lungs in pneumonoconiosis show inflammatory swellings and induration as well as areas of atelectasis and swelling of the bronchial glands with enlargement of the hilus shadows. Thus, in pneumonoconiosis, there can arise pictures very similar to submiliary and miliary tuberculosis except that in the former the shadows are larger, sharper and denser or more crowded than in tuberculosis."

The only journal article that I have been able to find after a rather thorough search of the literature appeared in the *Fortschritte auf dem Gebiete der Röntgenstrahlen*, 1915–16, XXIII, p. 19, by Michail Entin. He reports in detail five cases carefully studied in the Medical Clinic of the University of Basel from 1911 to 1914, three of these cases coming to autopsy.

Following are the reports on the roentgenograms of his five cases:

Case I.—Roentgenogram showed definite, not sharply outlined shadows in the upper half of both lungs varying in size from a ten-cent piece to a fifty-cent piece. The apices are not as markedly involved as the region between the second and the fifth ribs. The mottling is less marked than in the succeeding cases. The appearance was similar to that of tuberculosis. Autopsy report showed pneumonoconiosis of the upper lobes with no tuberculosis.

Case II.—Roentgenogram showed a rather equal mottling of both lungs from above downward. This is somewhat grosser than is seen in miliary tuberculosis, the individual shadows are less sharply outlined, the extreme apices are less extensively involved. The hilus shadows are markedly thickened. On the left side the shadows extend from the hilus toward the apex. About the hilus on both sides are irregular shadows about the size of a bean which consist of collections of small shadows. These are more marked on the left side. Patient left the hospital with the diagnosis of pneumonoconiosis.

Case III.—Roentgenogram showed sharply pronounced hilus shadows with moderately gross shadows equally distributed through both upper lobes. Lower lobes normal. Autopsy showed pneumonoconiosis; no tuberculosis.

Case IV.—Roentgenogram showed marked hilus shadows on the right side with narrow bands extending to the apex and to the lateral part of the diaphragm, with moderately fine mottling of the whole right lung, but especially marked in the mid portion. Beside the hilus are several rather sharply marked shadows; in the diaphragm two triangular points (adhesions). The left lung shows a similar picture, the mottling being especially marked in the upper and middle portions. Evidence of adhesions to left diaphragm. Patient died; no autopsy. Diagnosis, pneumonoconiosis.

Case V.—Roentgenogram showed a rather marked thickening in the upper half on both sides, produced by fine equally distributed shadows of dash and dot-like character. This is especially marked on the left side. Extreme apices not principally involved. From the upper half two shadows proceed to diaphragm (adhesions). Hilus shadows not especially marked. Patient died, no autopsy. Diagnosis, pneumonoconiosis.

Entin considers of importance in the differential diagnosis from chronic tuberculosis the following points:

In pneumonoconiosis, the most extensive involvement is apt to be in the subapical region.

Both sides of the lung are more evenly involved than in tuberculosis.

The fever is lower.

There are more marked symptoms of cardiac decompensation and more marked cyanosis.

Subjective dyspnea is more marked.

The roentgenogram shows an equal mottling best seen at each side of the hilus similar to that seen in miliary tuberculosis but somewhat larger and less sharply marked. In less typical cases the mottling is not so uniformly distributed and is found only in spots. Where the mottling is not typical, we find rather a uniform distribution on both sides. One also notes that the size, intensity and form of the single spots are more uniform than in tuberculosis. The markings extending out from the hilus are more uniform than in tuberculosis.

Entin summarizes his conclusions by stating that the *x*-ray appearance is characterized by a rather equal mottling of the lungs similar to miliary tuberculosis except that the spots are larger and less sharp. In the typical cases, the spots are of equal size on both sides.

Rieder, Entin and Barker all agree that the condition closely resembles miliary tuberculosis on the roentgen plate, but that in typical cases the shadows are somewhat larger, more uniform in size and more even in distribution than are the shadows in miliary tuberculosis. But what of the atypical cases?

Among the articles on the roentgenographic diagnosis of pulmonary tuberculosis, I have found no mention of pneumonoconiosis although, as we have seen, in the typical instances the picture resembles that of miliary tuberculosis rather closely and, if we consider the pathology, it might in the less advanced cases be expected to give an appearance rather closely simulating that described in tuberculosis by Dunham and myself in 1910 and since emphasized by Dunham, except that in pneumonoconiosis the distribution should be more general than in tuberculosis.

The following statements by Dunham are of interest in this connection:

"One of the most striking characteristics of the tuberculosis picture is the varying degree of change in the different groups of trunks in contrast to the general change of other diseases which might simulate tuberculosis."

And again, "there are many non-tuberculous pulmonary lesions, but we will not at this time discuss the stereoroentgenographic appearance of acute pneumonia, gangrene, cyst, etc., except to say that they should not be difficult to differentiate from tuberculosis. There is, however, a large class of cases clinically and roentgenographically suggesting pulmonary tuberculosis which, upon examination, disclose interesting and puzzling appearances. The hilus shadows and heavy trunks are increased in area and density; the linear markings are sharply defined, radiate, and often reach close to the periphery. These changes are often diffuse rather than localized and there is also an absence of the interweaving and studding characteristics of the tuberculosis cases."

Is this not a description of the milder cases of pneumonoconiosis such as occur in city dwellers and especially in those engaged in more or less dusty occupations?

In the light of the foregoing review of the literature, the following three cases are of interest and I am reporting them in the hope of stimulating further study on this subject.

CASE I.—Mr. W. R. (No. 47578), age 56, a Scotch laborer, presented himself at the Stanford University Medical Clinic, Oct. 15, 1916, complaining of asthma of four years' duration.

Family history unimportant.

Past History.—General health good. "Typho-pneumonia" five years ago. Malaria every summer for the past four years. No other acute diseases. Respiratory system negative except for some shortness of breath. Circulatory, digestive and urinary systems negative.

Sexual.—Denies lues. Neisser infection several times as a young man.

Residence and Occupation.—Born in Scotland. Worked in the coal mines of Pennsylvania from the age of 8 to 25. Since then has lived on the Pacific Coast, doing shipyard, railroad and tunnel work.

Habits good.

Present Illness.—Four years ago, after exposure, became very short of breath, so much so that he had to sit up for comfort. Had the desire to cough, but cough was unproductive. Since this onset he has had more or less constant dyspnea, but after exposure to wet or cold he is apt to be taken with a severe attack of dyspnea accompanied by pain in the chest. These attacks have been becoming more frequent, more severe and of longer duration. In none of these does he cough or expectorate, although he feels that to do so would give him relief. Lost 30 lbs. in weight.

Physical Examination by Dr. Hewlett.—Patient sallow. Mucous membranes pale and somewhat cyanotic. Pupils equal, reacting to light and accommodation. Throat negative.

The chest markedly barrel-shaped. Respirations labored with retraction of lower ribs and interspaces. Backs hyperresonant. Lower borders of lung unusually low. Fremitus negative. Expiration very prolonged with numerous crackling râles at end of inspiration, especially over the lower lobes. A few musical râles also heard. The fronts hyperresonant, especially the left apex. There is some impairment beneath the upper sternum. Breath sounds and râles the same in front as over the backs.

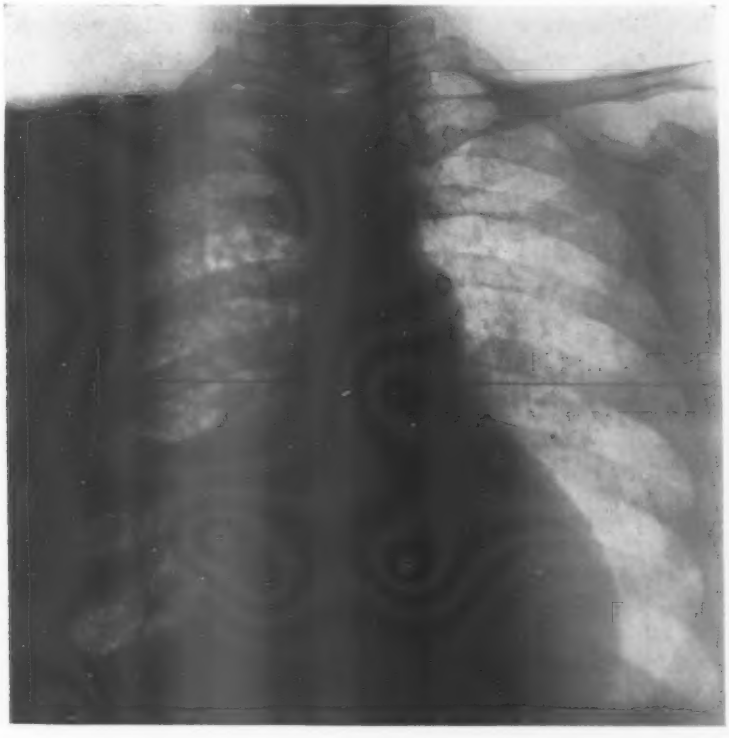

Fig. 1.

Cardiac apex seen in the fifth intercostal space just inside the mammary line, 9 cm to left of midline. Heart sounds clear. P_2 accentuated; radial arteries sclerotic.

Blood pressure 178–110.

Genitalia negative.

Reflexes normal.

Temperature (rectal) varied between 97.8 and 99 except for a rise to 100 following a sinus examination.

Pulse varied between 120 and 85.

Respiration 20 to 45. There was a suggestion of the Cheyne-Stokes' type except that there was no definite period of apnea.

Urine.—Faint trace of albumin; no casts seen.

Blood.—Reds 5,272,000; Hb. 86%; whites 8,280; polymorphonuclears 60%, lymphocytes 35%, large mononuclears 1%, eosinophiles 1%, basophiles 2%, transitionals 1%.

The stool showed no abnormality.

No sputum present. Patient had no expectoration.

Wassermann test, negative.

Roentgenogram of chest (Fig. 1) shows a diffuse mottling of both lungs least marked at apices. The dense areas are fairly uniform in size and are about the size of a large shot. Hilus shadows not especially thickened; no large areas of consolidation. Heart somewhat enlarged in transverse diameter. The appearance suggests a miliary tuberculosis except that the areas are somewhat larger and more uniform in size and distribution than those seen in miliary tuberculosis.

A careful consideration of all the findings in this case led to the diagnosis of pneumonoconiosis. At the last report the patient's condition was unchanged. We hope to be able to follow this case.

Case II.—Mr. C. (No. 46792), a Spanish miner aged 30, presented himself at the Stanford University Medical Clinic, Oct. 31, 1916, complaining of pains in his chest and marked shortness of breath for the past three months, loss of weight and strength for the past seven months.

Family history unimportant.

Past History.—Patient has worked in the mines of Spain, and for the past five years has worked in a copper mine in Shasta County where he has been constantly exposed to dust and gas.

Present Illness.—Patient well up to seven months ago, when after exposure he contracted a severe cold but with little cough or expectoration. This so-called cold lasted but a short time, but there remained a general weakness and a steady loss of weight. Three months ago began to have pains in chest, apparently pleural. Marked dyspnea developed; slight cough and expectoration recently. Patient has lost 20 lbs. in weight.

Physical Examination by Dr. Hewlett.—Patiently markedly and persistently dyspneic; chest behind slightly fuller on the right than on the left. Sides move equally. Fremitus more marked on the left than on the right throughout back. Right upper more resonant than left upper, the latter being somewhat impaired. Similar difference but less marked over lowers. Both lower margins are low, the right being the lower of the two, extending to the costal margin. On both sides, there is retraction of the interspaces on inspiration, this being more marked on the right side. Throughout the right back the breath sounds are less loud than on the left. Over the left there are everywhere sharp, medium-sized râles. Whispered voice sounds diminished over right. Bronchovesicular respiration near angle of left scapula. There is no succussion. Coin-sound not definitely obtained.

In front, the right chest is fuller than the left. Both clavicles are prominent. Respiration is mostly abdominal. Chest is in maximum inspiration. Fremitus is more marked throughout the left. Percussion is lower pitched over right front. Sounds are everywhere louder on the left with numerous crackles.

P. M. I. is well seen in fifth intercostal space just within the nipple line. Sounds clear, heart rapid but regular.

Temperature maximum 100.5° F. (rectal) after sinus examination.

Pulse ranged between 75 and 120.

Respiration 20 to 50 per minute.

Urine test shows a few granular and hyaline casts.

Sputum examination showed no tubercle bacilli on repeated test.

Blood.—Reds 5,984,000; Hb. 90%; whites 17,800; polymorphonuclears 76%; lymphocytes 26%; eosinophiles 2%.

Stool.—No abnormality.

Wassermann negative.

Roentgenogram of the chest (Fig. 2) showed marked mottling of left lung field most marked in mid portion, the mottling consisting of many rather sharply defined areas about the size of an ordinary shot, but in some cases being so dense as to cause a uniform clouding. The dome of the left diaphragm is peaked, and from the apex a dense shadow extends upward toward the hilus. The apical region is not densely infiltrated.

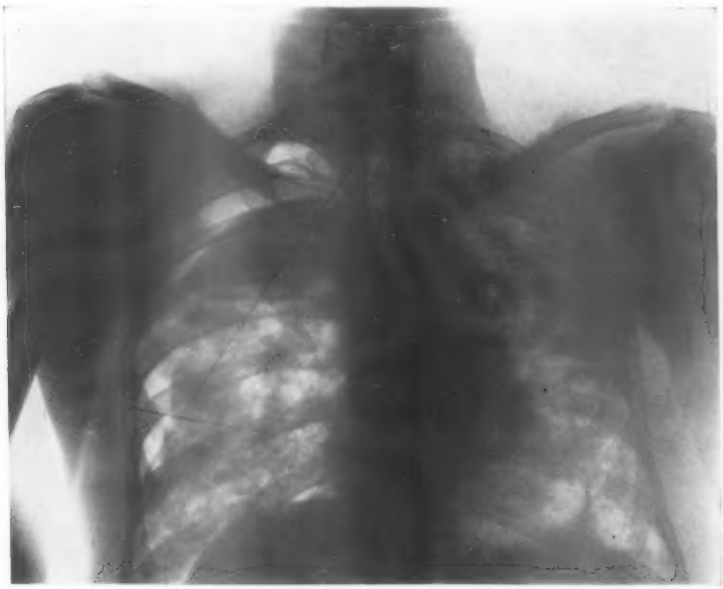

FIG. 2.

On the right side there is a definite retraction of the lung from the chest wall most marked in the upper part and decreasing toward the base. The upper portion of the retracted lung shows marked uniform increase in density; below is the same mottling as noted on the left side. Superior mediastinal shadow somewhat widened.

With no hesitation, the diagnosis on these plates was given as marked tuberculosis with pneumothorax on the right side. However, consideration of the history and physical findings led to the diagnosis of pneumonoconiosis, possibly associated with a tuberculosis.

During the night of October 14, 1916, the patient became weaker. Pulse rapidly failed and patient expired with picture of cardiac failure.

We were unable to obtain a post-mortem on this case but feel that we are justified in reporting it especially when taken in connection with the next case, which occurred in the service of Dr. Howard E. Ruggles. He has very kindly furnished me with the complete reports.

CASE III.—Mr. M., an Italian miner, age 64, entered St. Luke's Hospital May 1, 1914, complaining of shortness of breath of two months' duration.

Family history unimportant.

Past history unimportant except for pneumonia at the age of 25, and the fact that he had been a miner all his life working in copper, gold, silver and coal where he had been constantly exposed to dust.

Present Illness.—On March 1, 1914, two months before admission, patient was taken with severe pain in the left side of chest which disappeared in 24 hours. Three

days later had a similar attack of pain in the right chest persisting for 36 hours. With this attack the patient noticed that he was short of breath. This persisted. He had no chills or evident fever.

Examination on admission to hospital showed some impairment of the right side and of the left upper with vesicular breath sounds and no râles.

Temperature showed no elevation.

Pulse ranged in the neighborhood of 80.

Respiration 20–26.

Urine.—No abnormality.

Examination at the time of his readmission showed retraction of the infra- and supraclavicular spaces with marked retraction of the intercostal spaces especially on inspiration; absolute dullness of apices; right side less resonant than left. Posteriorly there was dullness over both apices shading into hyperresonance in the bases; bronchial and bronchovesicular breathing over these impaired areas with many large and small crackling râles; fremitus increased.

No elevation of temperature.

FIG. 3.

Blood.—Hb. 85%; whites 9,600; polymorphonuclears 70%; large mononuclears 4%; small mononuclears 15%.

Sputum.—Repeated examination showed no tubercle bacilli.

Roentgen examination (Fig. 3) showed marked infiltration throughout right lung and upper half of left lung; left base clear.

Patient's stay was uneventful and with rest his general condition improved somewhat so that he was discharged June 9, 1914.

On Feb. 12, 1915, patient re-entered hospital. After his discharge in June, he gradually became weaker, with slight cough, no expectoration, increasing dyspnea and no apparent temperature.

Pulse 80 to 120.

Respirations 30 to 44.

Urine again negative.

Blood count showed 12,000 whites.

Sputum.—No tubercle bacilli on repeated examination.

Patient remained in hospital, gradually failing, and died March 1, 1915.

Autopsy by Dr. Knapp showed both lungs very black of a leathery-like consistency on section. Sections show a very extensive peribronchial and septal fibrous thickening in which are diffuse deposits of black pigment. Very many of the alveolar spaces are partly filled with desquamated epithelium containing much intracellular pigment of the same jet black appearance.

There is very little cell reaction about any of the deposits but in the denser fibrous areas there is much necrosis and hyaline change. Diagnosis: pneumonoconiosis.

The *x*-ray appearance in this case was so striking that there was no doubt in the minds of those who had seen the plates that it was one of advanced chronic fibroid tuberculosis. Yet, a careful consideration of the history and clinical course suggested the possibility of pneumonoconiosis and this was confirmed at autopsy.

SUMMARY

It would seem from this review that the cases of pulmonary fibrosis resulting from chronic dust inhalation might be divided into three groups; the first to include at least some of the cases of diffuse and uniform thickening of the linear markings and trunks so well described by Dunham and with which every roentgenologist is familiar. These are indeed difficult cases for diagnosis. Of the points of differentiation above referred to by Dunham, the uniform character and distribution of the process would seem to be the most valuable. The absence of studding is of doubtful value, since nodular areas are produced in pneumonoconiosis which, from their character and location, must resemble the "studdings" of pulmonary tuberculosis. The absence of "interweaving" seems also of doubtful value in differentiation, but further study controlled by post-mortem examination will be necessary to settle this point.

The second group would include the so-called typical cases (Case I), which should be quite readily recognized if one bears in mind the uniform size and even distribution of the areas of increased density as pointed out by Entin, Rieder and Barker.

In the third group, we have the cases with marked and extensive pulmonary infiltration such as was present in Cases II and III of this series. These are the most difficult of differentiation. The shadows are too dense to allow satisfactory stereoscopic study and no individual areas are distinguishable owing to the density of the process. Differential diagnosis here must rest on a careful study of the history and the clinical course of the disease, but absolute diagnosis cannot be made during life. One feature of the roentgenogram in this type, which may be of help, is the apparent uniformity of the stage of the process, no area appearing more advanced than another.

It is to be hoped that we will have further study of this subject with post-mortem control, but it seems quite definite that chronic dust inhalation may produce a pulmonary fibrosis that results in a roentgenographic picture closely simulating early tuberculosis, miliary tuberculosis or advanced fibroid tuberculosis.

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THE TREATMENT OF HYPERTHYROIDISM*

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MUCH has been written in the last few years about various methods of treatment of goitre. Well-known internists have published reports of a large number of goitre cases which have been cured by medical treatment alone, while equally well-known surgeons profess that the propriety of universal surgical intervention in these cases is now practically conceded. There are the advocates of pet methods of physical therapy, injections of hot water, massage of greater or less violence, percussion of the cervical vertebræ, application of high frequency currents, etc., and last but not least the use of the roentgen ray.

As is the case in all instances where competent men disagree on a subject, there is some truth in the apparently differing opinions of all. The sensible conclusion to draw in the subject under discussion is that all are right in part and that most cases of hyperthyroidism may be treated by some one of these methods. The next logical step in our line of reasoning is that there must be a preferable treatment in each case.

There are what I believe to be general indications for the adoption of a treatment, although the individual circumstances of a case may alter the rule, and it is the object of this paper to define and classify such indications. The result can probably not lay claim to being exhaustive or systematic, but as a working basis from a therapeutic standpoint it seems to me to be a satisfactory foundation. The cases of hyperthyroidism which come to our attention may be divided into four groups: (1) Simple hyperthyroidism; (2) acute exophthalmic goitre; (3) chronic exophthalmic goitre; (4) hyperthyroidism developing on an old goitre, either hypertrophic or cystic. The first three groups of the classification are probably different stages of the same pathological condition, although we have never been able to absolutely satisfy ourselves on this point.

(1) *Simple Hyperthyroidism.*—By simple hyperthyroidism is understood a persistent exaggeration of the physiological hyperthyroidism which occurs at adolescence and is occasionally seen in adult life during the menstrual period or during pregnancy. The majority of my own cases have been in young girls on the verge of womanhood, and represent a prolongation of the hyperactivity of the thyroid from puberty. We believe that the vast majority of these cases will recover spontaneously or by the aid of proper medical treatment. However, it is not wise to allow these cases to go without treatment since a fair proportion of cases of exophthalmic goitre have their origin in the hyperthyroidism of puberty. We believe that operation in these cases is not only unnecessary but absolutely contraindicated. In our practice we have had 100% of cures in this type of hyperthyroidism by roentgen treatment.

(2) *Acute Exophthalmic Goitre.*—Under this heading we include all those cases of Basedow's disease in which the patient is acutely ill and in which the symptoms are becoming gradually but progressively worse. These are the classical cases of exophthalmic goitre, which often come on suddenly after fright or other nervous strain and in which the well-known clinical picture is present: tachycardia, palpitation, nervousness, tremor, weakness, sweating, headache, loss of weight, digestive dis-

* Read before the Pittsburgh College of Physicians, March, 15, 1917.

turbances and more or less pronounced eye symptoms. Enlargement of the thyroid is usually present but not necessarily so. If present, the swelling is soft and not hard as in some other types of goitre. If these patients are allowed to go untreated they either die or merge into what is called

(3) *Chronic Exophthalmic Goitre.*—When these cases present themselves, there is practically always an enlargement of the thyroid either of one or both lobes or of the entire gland. The swelling is much harder than in the acute type, the condition is more or less stationary, and the subjective pathological symptoms are much less pronounced. There are always tachycardia, tremor and nervousness, and usually also exophthalmos. The patient may not be losing weight, the appetite may be fair and the minor symptoms of Basedow's may be absent. These cases will continue in the same general condition for a long time.

(4) The condition of *hyperthyroidism developing on an old goitre* needs no explanation. It is evidently not a very common condition, as we have seen only two such cases in a series of sixty thyroid cases. The hyperthyroidism is readily amenable to roentgen treatment although the goitre itself remains practically unaffected, especially if cystic. The proper treatment for these cases is surgical.

The experience of the writer in the roentgen treatment of hyperthyroidism extends over a period of eleven years, and upon that experience the opinions and impressions advanced in this paper are based. However, the statistics quoted cover only the last four years, during which time 63 cases of thyroid disease have been treated in our laboratory (Drs. Johnston and Grier).

The two cases of hyperthyroidism on a basis of cystic goitre are understood to be cured of their hyperthyroidism only, while the cystic goitre remained. The cases of malignant thyroid and of persistent thymus in infants do not really belong in this table and are omitted in computing the total percentage of cures. The two cases of thymus in adults are also omitted from this computation. These two cases probably belong in the classification of exophthalmic goitre complicated by thymus, as some of the typical symptoms of thyrotoxemia were present. However, since the presence of thyroid disturbance was not definitely substantiated we present these cases separately.

TABLE OF CASES TREATED IN THE ROENTGEN LABORATORY OF DRS. JOHNSTON AND GRIER DURING FOUR YEARS ENDING MARCH 1, 1917

	Cases	Cured	Improved	Stopped treatment	Died	Operated	Still under treatment
Simple hyperthyroidism	8	6	—	—	—	—	2
Acute exophthalmic goitre	19	14	—	1	—	1	3
Chronic exophthalmic goitre	19	13	3	—	—	—	3
Exoph. goitre complicated by thymus	3	3	—	—	—	—	—
Exoph. goitre complicated by pregnancy	3	1	1	—	—	—	1
Hyperthyroidism from substernal thyroid	3	3	—	—	—	—	—
Hyperthyroidism on old cystic goitre	2	2	—	—	—	—	—
Malignant thyroid after operation	1	1	—	—	—	—	—
Malignant thyroid without operation	1	1	—	—	—	—	—
Thymus							
Infants	2	1	—	—	1	—	—
Adults	2	—	2	—	—	—	—
Total	63	45	6	1	1	1	9
Total cases of hyperthyroidism discharged	48	42	4	1	—	1	cured 87.5%

The two cases of malignant thyroid have stayed cured for three years.

The cases of exophthalmic goitre complicated by thymus were of the type designated as acute exophthalmic goitre, as were also the three cases of substernal thyroid. If these six cases are added to the 16 cases of acute exophthalmic goitre which have finished treatment, there are of this type 22 cases treated, with 20 cases discharged as cured or 90%. Referring to the table we find most favorable results in the following order: Simple hyperthyroidism, acute exophthalmic goitre, chronic exophthalmic goitre. This means that hyperthyroid cases respond to roentgen treatment according to the length of time the disease has been present, and not according to the severity of the symptoms. This

is hardly what one would expect. The severe case of acute exophthalmic goitre with very rapid pulse, extreme nervousness, emaciation, prostration, etc., would look to be the most unfavorable case for any kind of treatment.

The manner in which these cases improve under proper roentgen irradiation is almost miraculous. In fact one might say (within limits) that the more severe the symptoms, the more prompt will be the recovery. This is very gratifying, especially since these cases are bad operative risks.

The two factors which appear to have the greatest bearing on the prognosis under roentgen treatment are the length of time the condition has existed and the softness of the enlargement. Speaking generally, the longer the disease has been present and the harder the gland, the greater will be the difficulty in producing a satisfactory result. The same factors also determine largely the length of time the patient will require to be under treatment. Soft goitres which have only been present about a year usually recover in two or three months. Hard goitres of long standing seldom show much improvement inside of six months.

We do not believe it advisable and have never attempted to hurry the roentgen treatment of exophthalmic goitre. The massive dose method of treatment, especially in the acute type, is decidedly dangerous. The large amount of thyrotoxins thrown into the system from the rapid destruction of thyroid tissue is often alarming and might be fatal.

Under roentgen treatment the first improvement is noted in the nervousness and in the pulse rate. This is followed by an increase in weight and gradual amelioration of the other symptoms. If the tumefaction is soft, it will gradually decrease in size until it finally disappears. If hard, the rate of decrease will be much slower and chances of total disappearance much less.

In the early stages of the disease the exophthalmos is largely due to engorgement of the blood vessels of the orbit, the result of vasomotor disturbances. If treated at this time, the exophthalmos will disappear. Later in the disease, there is a deposition of fat in the orbit. Exophthalmos from this cause will not be influenced by roentgen treatment.

In regard to technic, part of the cases reported were treated with gas tubes, with small frequently repeated doses. Since the advent of the Coolidge tube all our treatment work has been done with the latter. Although the doses have been larger and less frequent, we do not use the maximum dose in these cases. No attempt was made in tabulating the cases to separate the cases treated by Coolidge and by gas tubes. Our impression is that there is little or no difference in the final result.

We have treated four cases after a ligation operation had been done. There was no improvement after the operation in any of these cases. Two recovered under roentgen treatment and two are still under treatment. The two under treatment are so much improved that we have no doubt of their final recovery.

We have treated several cases after extirpation of one lobe, also one case after extirpation of both lobes at two different operations. This case is very instructive and will bear a few words of elaboration.

Miss M., nurse, aged 28, suffering from typical exophthalmic goitre with enlargement of both lobes of the thyroid gland. One lobe was removed but the patient not getting any better, a second operation was performed and the other lobe removed. There being no improvement in the patient's condition after a few months, she came under our care. A fluoroscopic examination was made and a mass detected in the mediastinum. The mediastinum was irradiated by the cross fire method and after a surprisingly small number of treatments she completely recovered and has remained well since (about 18 months).

After this experience we have made a roentgen examination of all thyroid cases previous to treatment. This would also be a good procedure where operation is contemplated for removal of the thyroid.

The treatment of choice in hyperthyroidism we believe to be substantially as follows: The mild cases we have called simple hyperthyroidism may be treated medically or by the roentgen ray. Medical treatment will be successful in the majority of these cases; roentgen treatment will be satisfactory in practically all of them. Operation is unnecessary and improper in this type. In acute exophthalmic goitre roentgen treatment is the most satisfactory. Medical treatment is inadequate and surgery unnecessary. Furthermore operation in these cases is often dangerous. In chronic exophthalmic goitre the choice lies equally between roentgenization and operation. The roentgen treatment of these cases is apt to be slow but there is not so much danger in producing a subsequent hyperthyroidism as by operation. It should also be remembered that the death rate due to roentgen treatment as a procedure is nil. The final choice of treatment in this type will depend largely upon individual circumstances in each case.

Where hyperthyroidism develops in an old goitre, roentgen treatment is only palliative. The danger of producing myxedema is also greater here, and the proper treatment for these cases is surgical.

THE ACTION OF X-RAYS ON PLATE, PASTILLE AND SKIN

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THE photographic method of measurement of *x*-ray intensity as outlined by Dr. J. S. Shearer gives an accurate means of determining the variations by altering any of the factors. His formula is as follows:

$$\frac{\text{Current} \times \text{Voltage} \times \text{Voltage} \times \text{Time}}{\text{Distance} \times \text{Distance}}$$

For convenience suppose the following formula be taken:

$$\text{No. 1.} \quad \frac{20 \times 5 \times 5 \times 4}{20 \times 20} = \frac{2000}{400} = 5$$

If voltage 5 be doubled, the penetration is 4 times the above formula thus:

$$\text{No. 2.} \quad \frac{20 \times 10 \times 10 \times 4}{20 \times 20} = \frac{8000}{400} = 20$$

$20 \div 5 = 4$ times No. 1

If the plate is placed at half distance (10 in.), it will receive 4 times the amount of ray than in the original formula thus:

$$\text{No. 3.} \quad \frac{20 \times 5 \times 5 \times 4}{10 \times 10} = \frac{2000}{100} = 20$$

$20 \div 5 = 4$ times No. 1.

Therefore doubling the spark gap or reducing the distance one-half gives the same result on the plate.

If the milliamperage is doubled, the intensity is doubled, thus:

$$\text{No. 4.} \quad \frac{40 \times 5 \times 5 \times 4}{20 = 20} = \frac{4000}{400} = 10$$

$10 \div 5 = 2$ times No. 1.

If the time is doubled, we will get twice the intensity, thus:

$$\text{No. 5.}\quad \frac{20 \times 5 \times 5 \times 8}{20 \times 20} = \frac{4000}{400} = 10$$

$10 \div 5 = 2$ times No. 1.

Supposing one wished to duplicate the results obtained in using formula No. 1 on another plate but wished to obtain this in 1 second instead of 4 seconds: what voltage would be necessary?

$$\frac{20 \times KV \times KV \times 1}{20 \times 20} = \frac{20KV^2}{400} = \frac{KV^2}{20}$$

$$5 \div \frac{KV^2}{20} = 5 \times \frac{20}{KV^2} = \frac{100}{KV^2}$$

$$KV^2 = 100$$

$$KV = 10 \text{ volts}$$

How much milliamperage would be required to obtain the same result in 1 second instead of 4 seconds?

$$\frac{MA \times 5 \times 5 \times 1}{20 \times 20} = \frac{25}{400} = \frac{1}{16}$$

$$5 \times \frac{16}{1} = 80 \text{ milliamperes.}$$

How much time would be required to obtain the same result, if the distance were 25 in. instead of 20 in.?

$$\frac{20 \times 5 \times 5 \times T}{25 \times 25} = \frac{500}{625} = \frac{4}{5}$$

$$5 \times \frac{5}{4} = \frac{25}{4} = 6\tfrac{1}{4} \text{ sec.}$$

In the above calculations the factor of density of tissues is not taken into consideration. Supposing some one else is using an entirely different formula from the one you are accustomed to use in certain cases, and you wish to determine how these two formulæ compare from the standpoint of intensity and the contrast of the plates, proceed as follows.

For example:

$$\frac{30 \times 4 \times 4 \times 5}{18 \times 18} = \frac{2400}{324}$$

$\frac{2400}{324} \times \frac{1}{5} = 1\frac{39}{81}$ times more rays reaching the plate than with formula No. 1. Note that none of the factors are the same as in No. 1.

It must therefore follow that with a given formula of known intensity by the evidence on the *x*-ray plate, one can duplicate this by using other factors provided always that each factor is constantly maintained throughout the exposure.

PASTILLE DOSAGE COMPARED WITH ROENTGENOGRAPHIC PENETRATION

The foregoing formulæ and calculations on intensity for duplicating *x*-ray plates give a good idea of the part taken by each factor in producing constant results. When, however, this rule is applied to *x*-ray dosage from the standpoint of pastille dosage, we find that by doubling the spark gap instead of the pastille receiving four times the dose, as might be inferred from roentgenographic formulas, it receives but *twice* the dose.

To prove this statement the following experiments were made:

	MA	Sp.G.	D.	T.	P.	
1.—	3	3	8	2	½H skin	= 2H.
2.—	3	6	8	2	1H skin	= 4H.
3.—	3	9	8	2	1½H skin	= 6H.

MA = milliamperage.
Sp.G. = spark gap.
D = distance in inches from the target.
T = time in minutes.
P = pastille reading on Holzknecht radiometer when pastille is placed on the skin. The last column is the regular Holzknecht reading at half distance.

As a check on the above experiments the following factors were taken:

	MA	Sp.G.	D	T	P	
4.—	3	6	16	2	¼H skin	= 1H.
5.—	3	6	16	4	½H skin	= 2H.

By comparing experiments 4 and 2 it is obvious that the intensity of the ray is inversely as the square of the distance (distance being doubled, other factors remaining constant); but by comparing Nos. 4 and 1 it is seen that two factors, namely voltage and distance, are both doubled.

If the distance in No. 1 alone were doubled the pastille reading would be $\frac{1}{8}$ skin $= \frac{1}{2}$ H.

If the roentgenographic rule applied and the distance and spark gap in No. 1 were both doubled as in No. 4, the reading of

the pastille would be the same in both instances.

If the milliamperage is doubled, other factors remaining constant, the reading of the pastille is *doubled* because the quantity of ray is doubled; this corresponds exactly to what one would expect to find in a roentgenographic plate.

Experiment:

MA	Sp.G.	D	T	P
3	3	8	2	½ skin = 2H.
6	3	8	2	1 skin = 4H.

PLATE EXPOSURE AND SKIN TOLERANCE IN ROENTGENOGRAPHY

Doubling the voltage or spark gap in roentgenographic work, other factors remaining constant, gives four times the intensity on the plate. Dr. Shearer's formula follows:

1. $\frac{MA \times KV \times KV \times T}{D \times D}$ $\frac{20 \times 5 \times 5 \times 4}{20 \times 20}$

In unfiltered roentgenotherapy doubling the spark gap, other factors remaining constant, gives two times the pastille reading. Therefore, the following formula:

2. $\frac{MA \times Sp.G. \times T}{D \times D}$ $\frac{3 \times 3 \times 5}{8 \times 8} = 1\frac{1}{4}$ skin = 5H.

No. 2 represents factors which will produce an erythema dose in unfiltered treatments. The practical application of this formula for the roentgenologist is illustrated as follows:

Supposing one wished to determine the number of plates that could be taken of a frontal sinus with patient in one position before the skin will show an erythema or an epilation be produced with the following factors:

MA	Sp.G.	D	T
25	5½	18 in.	10 sec.

This means that the plate is 18 inches from the target and in this instance the scalp is 10 inches from the anode. In order to find the pastille dosage the scalp will receive, the distance in the formula must be changed from 18 to 10 inches, thus:

MA	Sp.G.	D	T
25	5½	10 in.	10 sec. = ⅙ min.

$$\frac{25 \times 5\frac{1}{2} \times \frac{1}{6}}{10 \times 10} = \frac{\frac{275}{12}}{100} = \frac{11}{48}$$

Take the standard formula for an erythema dose:

$$\frac{3 \times 3 \times 5}{8 \times 8} = \frac{45}{64}$$

Divide the erythema dose by the dose for each exposure:

$$\frac{45}{64} \times \frac{48}{11} = \frac{135}{44} = 3\frac{3}{44}$$

Thus three plates with these factors would approach dangerously near an erythema and consequent epilation. By dividing the product of the factors of the standard formula by any exposure formula provided the skin distance is taken, a roentgenologist can calculate the number of plates that can be taken without approaching too closely the erythema dose.

FILTERED AND UNFILTERED ROENTGENOTHERAPY

The filter used in the following experiments consists of 3 mm of aluminum.

	MA	Sp.G.	D	T	P
1.—	4½	9	7	4	2 skin H = 8H.
2.—	4½	9	14	4	1 skin H = 4H.

In this experiment two pastilles were used, one at 7 in. distance and one at 14 in. from the target, both pastilles being exposed at the same time. It will be seen that instead of the pastille at the half distance reading four times the amount of full distance as in unfiltered experiments, it reads only twice the amount.

If the spark gap is doubled, all other factors remaining constant, the pastille gives double the reading.

MA	Sp.G.	D	T	P
4½	4½	7	4	1 skin H = 4H.
4½	9	7	4	2 skin H = 8H.

If the milliamperage is doubled, other factors remaining constant, the pastille gives double the reading.

MA	Sp.G.	D	T	P
4½	4½	7	4	1 skin H = 4H.
9	4½	7	4	2 skin H = 8H.

Hence the formula for filtered dosage:

$$\frac{\text{MA} \times \text{Sp.G.} \times \text{T}}{\text{Distance}}$$

$$\frac{4\frac{1}{2} \times 4\frac{1}{2} \times 4}{7} = 1 \text{ skin H} = 4\text{H}.$$

The above formula which gives a pastille reading of 1 skin H may be taken as a standard for filtered readings instead of the formula for unfiltered treatment.

The following experiments using 3 mm of aluminum as a filter were made:

Gap	MA	D	T	P
6	5	10	3 min. 51 sec.	= 1 skin unit = 4H.
6	5	10	7 " 42 "	= 1¼ " " = 5H.
6	5	10	11 " 33 "	= 1½ " " = 6H.
6	5	10	15 " 24 "	= 1¾ " " = 7H.
6	5	10	19 " 15 "	= 2 " " = 8H.

Gap	MA	D	T	P
7	5	10	3 min. 18 sec.	= 1 skin unit = 4H.
7	5	10	6 " 36 "	= 1½ " " = 6H.
7	5	10	9 " 54 "	= 1¾ " " = 7H.
7	5	10	13 " 12 "	= 2 " " = 8H.
7	5	10	16 " 30 "	= 2¼ " " = 9H.
7	5	10	19 " 48 "	= 2½ " " = 10H.
7	5	10	23 " 6 "	= 2¾ " " = 11H.
7	5	10	26 " 28 "	= 3 " " = 12H.

Gap	MA	D	T	P
8	5	10	2 min. 53 sec.	= 1 skin unit = 4H.
8	5	10	5 " 46 "	= 1½ " " = 6H.
8	5	10	8 " 39 "	= 2 " " = 8H.
8	5	10	11 " 32 "	= 2¼ " " = 9H.
8	5	10	14 " 25 "	= 2½ " " = 10H.
8	5	10	17 " 18 "	= 2¾ " " = 11H.
8	5	10	20 " 11 "	= 3 " " = 12H.

Gap	MA	D	T	P
9	5	10	2 min. 34 sec.	= 1 skin unit = 4H.
9	5	10	5 " 8 "	= 1½ " " = 6H.
9	5	10	7 " 42 "	= 2 " " = 8H.
9	5	10	10 " 16 "	= 2¼ " " = 9H.
9	5	10	12 " 50 "	= 2½ " " = 10H.
9	5	10	15 " 24 "	= 2¾ " " = 11H.
9	5	10	17 " 58 "	= 3 " " = 12H.

Gap	MA	D	T	P
10	5	10	2 min. 19 sec.	= 1 skin unit = 4H.
10	5	10	4 " 38 "	= 1½ " " = 6H.
10	5	10	6 " 57 "	= 2 " " = 8H.
10	5	10	9 " 16 "	= 2¼ " " = 9H.

The time factor in the above experiments to produce 1 skin unit was determined by the filtered formula previously referred to. The time is then increased in arithmetical progression. It will be seen that the pastille reading on a 6 in. gap, when time is doubled, reads 1¼ skin units instead of 2 skin units; with a 7-inch gap it reads 1½ skin units instead of two; and when three times the original time for 1 skin unit is used, the reading is 1¾ instead of 2. On 8, 9, and 10-inch spark gaps the readings do not change to one-quarter increase until 2 units have been obtained. In the above experiment it might be well to point out that the pastille reading previous to the change of increase of ¼ H skin unit is approximately one-eighth of a skin unit short of a full reading.

PASTILLE READINGS AND SKIN ERYTHEMA

The practical value of the unfiltered experiments will be apparent as soon as the ratio of pastille readings and the skin erythema is determined. To obtain this ratio the following experiment was made:

Four areas of a patient's back were treated with the following factors for each area:

No. 1. $\frac{3 \times 3 \times 5}{8 \times 8} = 1\frac{1}{4}$ skin unit = 5H.

No. 2. $\frac{3 \times 6 \times 2\frac{1}{2}}{8 \times 8} = 1\frac{1}{4}$ skin unit = 5H.

No. 3. $\frac{3 \times 4\frac{1}{2} \times 3\frac{1}{3}}{8 \times 8} = 1\frac{1}{4}$ skin unit = 5H.

No. 4. $\frac{3 \times 9 \times 1\frac{2}{3}}{8 \times 8} = 1\frac{1}{4}$ skin units = 5H.

The photograph of the patient, taken ten days after treatment, demonstrates that all the areas coincide.

The areas that correspond in intensity of erythema were produced in each instance by doubling the spark gap and using one half the time, other factors being constant. It therefore follows that if the spark gap is doubled and the time reduced one-half, the same degree of erythema will be produced, other factors remaining constant.

A 9-inch spark gap and 5 MA will produce an erythema of the skin with a pastille reading of 2½ skin units = 10 H using 3 mm of aluminum as a filter.

Unfiltered Dose

10 MA and 12 in. distance is maintained for each gap

$$\frac{3 \times 3 \times 4}{8 \times 8} = 1 \text{ skin unit}$$

Gap	*¼ Skin Unit*	*½ Skin Unit*	*¾ Skin Unit*	*1 Skin Unit*	*1¼ Skin Unit*
3	40 sec.	1 min. 21 sec.	2 min. 1 sec.	2 min. 42 sec.	3 min. 22 sec.
3½	35 "	1 " 9 "	1 " 44 "	2 " 19 "	2 " 54 "
4	30 "	1 "	1 " 31 "	2 " 1 "	2 " 32 "
4½	27 "	54 sec.	1 " 21 "	1 " 48 "	2 " 15 "
5	24 "	49 "	1 " 13 "	1 " 37 "	2 " 1 "
5½	22 "	44 "	1 " 6 "	1 " 28 "	1 " 50 "
6	20 "	40 "	1 "	1 " 21 "	1 " 41 "
6½	19 "	37 "	56 "	1 " 15 "	1 " 33 "
7	17 "	35 "	52 "	1 " 9 "	1 " 27 "
7½	16 "	32 "	49 "	1 " 5 "	1 " 21 "
8	15 "	30 "	46 "	1 "	1 " 16 "
8½	14 "	29 "	43 "	57 "	1 " 11 "
9	13 "	27 "	40 "	54 "	1 " 7 "

The four areas of erythema produced as is shown by the illustration indicate that the pastille coincides with the biological effect on the skin.

Therefore the spark as a factor in its effects on the skin and pastille is just one-half as active as on the Kienboeck strips and roentgenographic plate.

The spark gaps used, namely, 3 and 6 and 4½ and 9 inches cover a wide and practical range of usefulness in *x*-ray therapy. The Kienboeck strips and *x*-ray plates may indicate more accurately the amount of ray reaching the part exposed, but from the standpoint of therapy the biological effect is most important.

In order to change a Holzknecht reading to a Kienboeck, it is necessary to double the Holzknecht reading. The reason for this is evident since doubling the spark gap gives double the dose on the pastille, while doubling the spark gap gives four times the dose on a Kienboeck strip.

In filtered roentgenotherapy using 3 millimeters of aluminum the spark gap acts the same as in the unfiltered experiments above referred to. Unlike the unfiltered, the one-half distance doubles the dose instead of giving four times the dose on the pastille at 7 and 14 inches, respectively.

The Holzknecht radiometer was not originally intended as a scale of dosage in filtered treatments, yet it is used and depended on entirely in giving deep cross-fire treatments. The time factor as indicated in the filtered reading differs in its results on the pastille reading on the 6-, 7-, and 8-inch gaps. With a 6-inch gap, employing

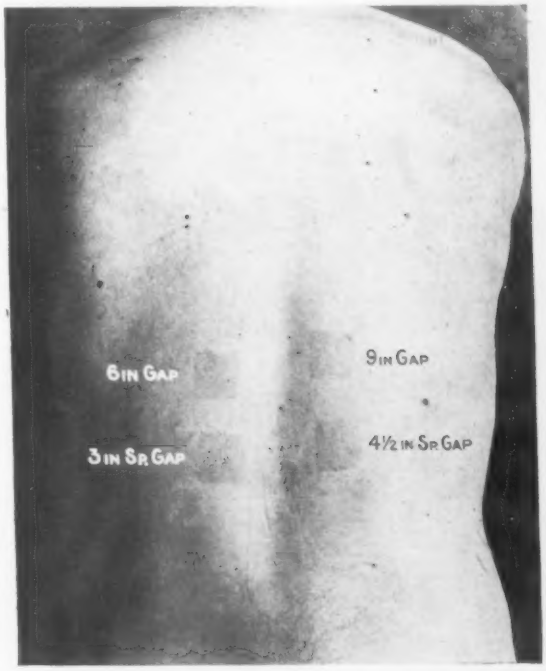


FIG. 1.

the 4 factors that will produce 1 skin unit or 4 H and then doubling the time, other factors remaining constant, a reading of 1¼ H or 5 H will be obtained. For each time this process is repeated the readings advance ¼ of a skin unit or 1 H. A 7-inch

gap gives 1½ skin units or 6 H when double the time to produce 1 skin unit as used, and then begins to advance at the rate of ¼ skin unit or 1 H. for each exposure time. The 8-, 9-, and 10-inch gaps with three times the time exposure for 1 skin unit gives 2 skin units and then begins to advance at the rate of ¼ skin unit for each exposure time.

Another method of determining the factors necessary to produce an erythema dose is by taking certain factors and exposing small areas of skin, giving each one varying intervals of time until one of the areas shows an erythema. These factors then may be taken as a standard for the individual equipment, and from this various doses may be determined by means of the formula, provided the factors are maintained constantly throughout the exposure.

To use the pastille radiometer method, one would find it necessary to work out two standards, one for filtered and one for unfiltered therapy, but the method above discussed obviates the necessity of the use of the pastille and radiometer.

The difficulties of carrying out such a method are at the present time greatly enhanced, owing to the lack of standardization of the various equipments now in common use.

SUMMARY

In unfiltered dosage, doubling the spark gap doubles the dose, other factors remaining constant; otherwise Dr. J. S. Shearer's roentgenographic rules apply.

In filtered dosage, doubling the spark doubles the dosage as in unfiltered, but doubling the distance gives one-half the dose instead of one-quarter, as in both unfiltered and roentgenographic formulæ.

Skin erythema of two areas are identical when the spark gap of the original is doubled and one-half the time is taken, other factors remaining constant.

AN UNUSUAL POSITION OF URINARY CALCULI

BY GEO. C. JOHNSTON, M.D., AND GEO. W. GRIER, M.D.

PITTSBURGH, PA.

THE patient in the unusual case we are going to describe was a 24-year old Greek who was referred by Dr. C. A. Hill for a roentgen examination of the genito-urinary tract. The subjective complaints consisted in colicky pains throughout the abdomen and pain centering in the region of the scapula.

Family history was negative. At the age of 7, "belly ache" was complained of, the pain being referred to the left kidney. This condition persisted until the age of 15, when blood appeared in the urine, accompanied by frequent severe pain. No vomiting.

The clinical examination showed normal liver and stomach, and tenderness on deep pressure at the left of the umbilicus. Blood and kidney examinations negative.

Roentgen Examination.—The roentgenogram showed three shadows just above the crest of the ilium, suggesting gallstones (Fig. 1), although it seemed hardly possible for the gallbladder to occupy that position.

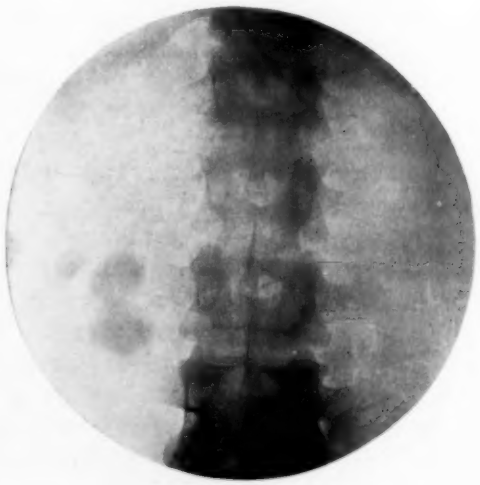


FIG. 1.

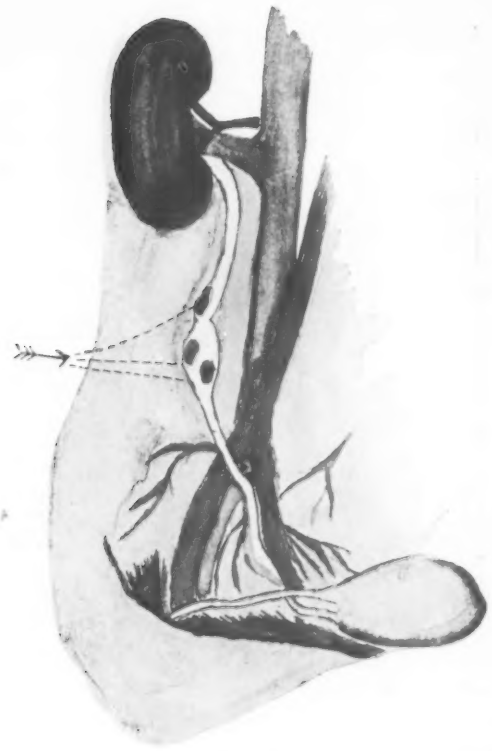


FIG. 2.

The position was also too low for the kidney, unless that organ was very much prolapsed. But as the condition occurred on the left side and prolapse of the kidney is unusual on that side, the weight of evidence was against the probability of renal calculi being the cause of the shadows. Being unable to say definitely whether the shadows were in the gallbladder or the kidney, the possibility of foreign bodies in the intestines was thought of, but when another roentgen plate was taken after the bowels had been cleansed out and the shadows again appeared in the same places, operation was advised.

FIG. 3.

At operation, two large stones were felt at about the middle of the left ureter and removed through an incision through the peritoneum, and then through the ureter. The ureter at this point was greatly dilated, and about two inches above there was a small stone incarcerated in a little pouch of the ureter (Fig. 2). The two large stones were faceted and articulated with each other after the manner of gallstones.

METHOD FOR DENTAL STEREOROENTGENOGRAPHY

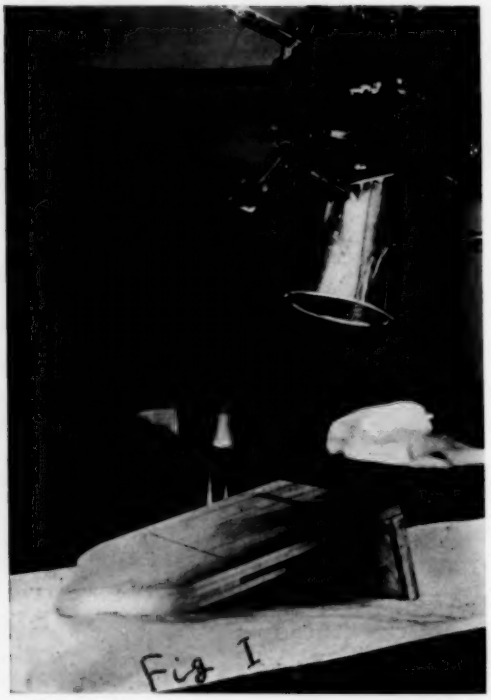
BY HENRI LETORD, D.D.S., AND CHARLES G. LUNAN

EL PASO, TEXAS

DENTAL roentgenography has generally been confined to the use of single roentgenograms as in multiple films or *x*-ray plates. Until recently the writers have been accustomed to making plate records of the upper and lower jaws, supplementing these, when necessary, by dental film records. No argument is necessary to convince one that the stereoroentgenogram is superior to a single plate in clearing up doubtful points.

Using as a model the old type of "frontal sinus" board, we had built a duplicate with a shallow tunnel underneath to accommodate a 6½ by 8½ inch plate. (Figs. 1 and 2.) These illustrations show more plainly than any written description the groove where the plate is inserted and the rectangle marked on the surface directly overlying the plate. Care is taken to shake the plates into similar positions in the envelope before inserting them.

The surface of the board measures 15½ inches in length and 11 inches in width.



The thickness is 1¼ inches. The back piece measures 11 inches by 3½ inches. The total length of the slot measures 7¼ inches, and begins 2 inches from the back of the instrument. There is a notch on the under surface to make it easy to withdraw the plates. The slot is one quarter of an inch in width.

We employ a tube stand of the Kelly model. Any good tube stand will answer, provided it permits easy manipulation of the tube carriage for stereoroentgenography.

The technic is very simple: First, make an exposure; second, change the exposed plate for a second plate, move the tube 2½ inches, and continue with the second exposure. It is important to see that the long edge of the rectangle, indicating the position of the plate, is parallel to the long supporting bars of the tube stand.

TO DR. WALTER J. DODD

Once more the sad, pathetic plaint goes forth,
Not this time from the far Judean plain,
But from the New World, with its surging life;
"He saved others, himself he cannot save."

Go lay your wreaths of laurel on his brow,
Go drop the heartfelt tears upon his grave;
'Tis all the outward tribute most of us can pay,
But in our inmost souls his place is dear.

Not at the battle front was thy fight won,
Not in the marts of noisy trade and strife,
But in the quiet chambers where the modern light
Of latest science did its wondrous work.

The call of duty was to thee a trumpet blast.
What though its doing was to thee thy doom!
No craven blood flowed in thy veins, thou hero grand!
All sacrifice was for thee a joy to make.

A willing martyr to the sacred cause
Of suffering mortals, racked with hopeless pain.
A Pioneer developing those marvelous *x*-rays
So full of promise for the hope of man.

What must have been thy welcome going home
To take thy place with the Immortals' Throng?
But, ah, what hast thou left behind? No tongue can tell,
Of honor, love and gratitude from all mankind.

EDWARD AXTELL.

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EDITORIALS

DEEP ROENTGENOTHERAPY

The abstract of the article by Wintz in this issue raises a question of the possibility of producing homogeneous rays, i.e., a beam in which there is only a small group of nearly equal wave lengths. The question of the value of such radiation can only be settled by experience and one may doubt whether the results will be of revolutionary character.

The author is mistaken in believing that it is a long way to such radiation. By using special metals for targets and suitable filters, a very high degree of homogeneity can be ensured, and a considerable quantity produced at the same time. Experiments on the use of this radiation may be expected very soon.

The introduction of a grounded plate inside the tube may serve to stabilize the tube action and would tend to reduce charges in the glass walls. The proposition to introduce such a grounded tube into the body is likely to be very dangerous.

Grounding does not reduce the voltage between terminals and the tendency to arc from either terminal to the body will be great. A metal tube such as shown by Dr. Coolidge at Chicago could be made safe, if perfected for this work.

J. S. S.

DIAGNOSIS OF INTESTINAL STASIS

It is gratifying to note from recent contributions to the medical literature that roentgenological examination of intestinal conditions for the purpose of diagnosing intestinal stasis is becoming more generally recognized in the routine practice of well known internists. Perhaps it is a more profitable field of endeavor than resorting to major operations for minor ailments, inasmuch as a roentgen examination will furnish valuable information in a large number of cases in regard to the functional and physiological activities of the individual bowel in addition to showing up kinks and angulations which may be physiological and not be the cause of the underlying complaint.

So far as the stomach is concerned, there is hardly a dissenting voice now in regard to the importance and indeed necessity of roentgenological aid in the diag-

nosis, but as far as the intestinal tract is concerned, and particularly the conditions of the small intestine, the significance of this aid does not seem to have been appreciated as yet to the extent it deserves. But the tide is turning.

An "Alleged Invention" Exposed

Our readers will recall an editorial in this JOURNAL for November, 1916, signed by Dr. E. W. Caldwell, calling attention to the "alleged invention" of a man named Shearer who, at the time, was a member of the British Army Medical Corps. He was supposed to be an American, a graduate of a medical school in Washington, D. C. He claimed to have devised a new electrical method for the delineation of internal viscera.

From a physician serving with the allied armies who has had abundant opportunity to investigate the facts, we learn that this man was finally pronounced a fraud and that he is at the present time serving a term of imprisonment for fraud. Dr. Caldwell's estimate of the invention seems to have been justified by later developments.

Commercial Roentgen Laboratories

During the last two or three years, many commercial roentgen laboratories have sprung up in the larger cities.

By Commercial Laboratory is meant one which advertises to make "*X*-Ray Pictures" at so much per picture. The work in these laboratories is usually done by technicians. Interpretations of plates are made sometimes by an associated physician—licensed or unlicensed—and the important and indispensable aid of roentgenoscopy is usually ignored.

It is claimed that the Commercial Laboratory supplies a supposed need for cheaper roentgen diagnosis. The alluring circular of one of these laboratories announced that "*x*-ray diagnosis is now brought within the reach of everyone." The lure is made still stronger by an offer of 20 per cent commission to physicians whose patients are referred for the purchase of *x*-ray pictures.

According to the fee schedule of one of these advertising concerns the charges are: $5.00 for an 8x10 plate and $7.00 for a 14x17 plate. As an evidence of the quality of the "pictures," we are assured that the technician has had years of experience in well-known hospitals.

The appearance of these Commercial Laboratories brings up the old question of whether roentgen diagnosis is a medical specialty or whether is belongs in the realm of photography. The public and many of our alleged colleagues in medicine, care not the least about the question, if only they can buy cheap "*x*-ray pictures" and get the 20 per cent rebate.

The public cannot be blamed for desiring to obtain the best service possible for the money expended, and IF these Commercial Laboratories are, as they claim, producing reliable results for smaller fees than those charged by practicing roentgenologists, why should they not be encouraged? By the same token, why should we not lend our encouragement to commercial institutions which advertise skillful medical or surgical treatment at lower fees than the profession now charges?

The history of such commercial medical institutions has usually shown that their work is not reliable and that they do not save expense for the patient. It is reasonable to expect similar misrepresentations from Commercial Roentgen Laboratories and also from certain similarly managed hospital laboratories, where so-called "*x*-ray pictures" are sold at so much per picture according to a printed schedule.

The reputable roentgenologist does not charge so much per plate, but usually makes a fee for an examination, which includes roentgenoscopy and any number of plates that may be necessary. If the fees of the average conscientious roentgenologist were reduced to so much per plate, it would be found that the amount per

plate would be somewhat less than the charge per picture of the Commercial Laboratory. The claim that the Commercial Laboratory does the work cheaper, is true only when the examination is not made thoroughly and safely. It is therefore difficult to avoid the conclusion that these commercial picture shops, whether managed by hospitals or private individuals, are misrepresenting their wares and cheating their patients (?), clients, or customers.

Another objection to the Commercial Laboratory and to the hospital laboratories in which a false economy permits the work to be done by technicians, is that such institutions offer a competition which discourages competent medical men from taking up this medical specialty.

The field of roentgenology is large and the number of really competent men in it is far too small. The common impression that the financial returns from this work are large is prevalent only among those who know nothing about the expense connected with it. Although the *x*-ray is more than twenty years old, it would be difficult to find a roentgenologist whose earnings compare favorably those of men of similar standing in other medical specialties.

It seems likely that the competition of Commercial Laboratories and of hospitals employing technicians may keep out of the practice of roentgenology men of scientific training who might reasonably be expected to give reliable service and to continue the work of developing the art. There may be proper places for the technician in roentgenology, but it must be remembered that every Commercial Laboratory and every hospital depending solely upon technicians for its work is, to a certain extent, replacing men who may contribute to the development of the art with those from whom no advancement of the art has ever sprung.

We cannot hope entirely to eliminate the quack from the practice of medicine, but it is time to consider whether the quack in roentgenology should not receive the same attention from the Medical Societies that other quacks receive. Perhaps some amendment of the legal definition of the practice of medicine may be necessary. Those who have at heart the advancement of roentgenology as an aid to the healing art should discourage the peddling of so-called "*x*-ray pictures" either by hospitals or by those who, lacking the prestige of the hospital, must advertise their wares more blatantly.

E. W. C.

SOCIETY PROCEEDINGS

Chicago Roentgen Society

A regular monthly meeting was held on February 9, 1917, under the presidency of Dr. James T. Case.

Dr. Emil Beck presented a case of abdominal pregnancy in which the diagnosis had been well worked out by the roentgen evidence.

Dr. J. T. Case showed slides of a case of carcinoma of the sigmoid in which practically all of the physical symptoms had been referred to the region of the cecum and ascending colon, leading to a previous incorrect diagnosis of chronic appendix disease with adhesions in the ileocecal region. Dr. Case showed this instance to emphasize the frequency with which lesions of the pelvic colon give rise to symptoms in the right lower quadrant, leading to an erroneous diagnosis of cecal or appendical disease.

Mr. Montiford Morrison related a number of experiments being conducted by Dr. A. W. Hull of the Research Laboratories of the General Electric Company on the *X*-ray Spectrum of Tungsten. The complete description of these experiments will be found in the current volume of *British Archives of Radiology and Electrotherapeutics*.

Dr. Emil Beck concluded the evening by a paper on the "Differential Diagnosis

of Bone Lesions After Healing Has Taken Place." Dr. Beck illustrated by means of stereoroentgenograms the typical changes which take place in the bone during disease and after the disease has healed. Especially by the aid of the stereoscopic method post-tubercular, post-syphilitic and post-typhoid bone lesions show a striking contrast to the lesion of acute or active disease. Full discussion of the paper will later be made by Dr. Beck.

M. J. HUBENY, M.D., Sec'y.

SOCIÉTÉ DE RADIOLOGIE MÉDICALE DE FRANCE

E. Albert-Weil furnishes an account in *Paris Médical* for March 17, 1917, of the proceedings of the Société de radiologie médicale de France, a preliminary report of which has already appeared in the May number of this JOURNAL. The following is a translation of Dr. Weil's account.

The Société de radiologie médicale de France, which had suspended its meetings since the beginning of the war, held an extraordinary meeting on March 5, Justin Godart, under-secretary for public health, in the chair. The meeting was well attended.

At the time war was declared, the Public Health Service had practically nothing at its command except, perhaps, one or two roentgen cars. Since then great efforts have been made. Self-contained roentgen vehicles as well as roentgen automobiles connected with surgical ambulances have been constructed; great roentgen centres have been established in Paris and large provincial cities; and nearly all hospitals have established a temporary or auxiliary roentgen service. The organization at the disposal of the roentgenological fraternity was inadequate to keep all these arrangements in working order, and it became necessary to train physicians who had but little preliminary knowledge in this specialty. A body of assistants had to be added without any loss of time. All these endeavors culminated in a vast organization, for the conception and execution of which Major Haret, M.D., the secretary-general of the Société de radiologie and technical counselor to the war office, deserves the greater part of the credit.

After thirty months of war, the results achieved, the improvements still desirable, and the place where the roentgen rays belong in the service of the wounded, both in regard to diagnosis and treatment, were submitted to the Society. There were seven papers to be read. Ledoux-Lebard dealt with the extraction of projectiles with the aid of the roentgen rays, explaining the methods of applying the rays both anteriorly and posteriorly; Rechou detailed the ways of applying roentgen treatment in war injuries; Belot explained the exact localization of foreign bodies in the orbital region; A. Béclère spoke of the necessity of entrusting none but physicians with the direction of the roentgenological service; Aubourg and Barret went into particulars of the various types of installation and the rôle they would most usefully be assigned to for service in the field, and the same authors demanded in another paper that the roentgenologist should have a free hand to select his own method of examination in making a diagnosis; and Delherm reported on the results of using the roentgen rays in the diagnosis of pulmonary tuberculosis in soldiers.

Among all these communications that of Béclère is the most important. While doing justice to those physicians and others who, at the beginning of the war, were hardly qualified to undertake roentgenological work in its various forms, he showed that, valuable though their proffered aid was in 1914, it was wholly insufficient now that improvised measures had given way to organized effort. It had become an indisputable fact that the practice of roentgenology should be confined to physicians, as it was only physicians who could understand all that was useful for a diagnosis and who could correctly interpret the roentgen findings.

But the war had been instrumental in rekindling the imagination of those who believed that the handling of a roentgen tube and the taking of a roentgen picture constituted all there was in roentgenology. Béclère and many other physicians have proved from concrete examples that this is totally wrong, that it acts to the detriment of the wounded, that roentgenoscopy is superior to roentgenography, and that the operator should understand how to combine the advantages of both. It may seem to some that this is equivalent to forcing open doors, but I am one of those who think differently, and I hope that the Public Health Service will take seriously into account the ideas put forth by the eminent physician of the St. Antoine Hospital (Béclère).

Belot demonstrated with great lucidity the new method of localizing projectiles in the orbital region which he in collaboration with Fraudet had devised. With this method it is not only possible to say—what roentgenologists already know—whether a foreign body is movable with the substance of the eye but also whether the projectile has penetrated the globe of the muscles which control it.

Ledoux-Lebard explained the technical details of his method of extracting projectiles under the guidance of the fluorescent screen, while Rechou, in dealing with the same subject, showed the points in which his method differed from the former. The difference is principally this, that Ledoux confines himself to directing the surgeon from time to time, while Rechou takes the forceps himself and extracts the bullet after the first incision has been made by the surgeon under his directions according to the localization learned from the screen. To my way of thinking it will in most cases depend upon the understanding of the surgeon who is associated with the roentgenologist, as to which of these methods should be adopted in a given case.

Delherm's communication was in effect a demonstration of the truth of the point previously made by Béclère. Who else but a roentgen-trained physician, accustomed to the clinical difficulties of pulmonary affections, can utilize the roentgen rays to reveal initial tuberculosis, pleuritis, or to recognize that a number of symptoms which had erroneously been taken to be pulmonary, depend in reality on a pathological condition of the upper air passages?

I might say as much in regard to the interesting communication of Barret and Aubourg: none but a roentgen-trained physician can make a diagnosis, but in a great many instances he will succeed better with roentgenoscopy than with roentgenography, and that at less expense.

Rechou spoke authoritatively on the roentgen treatment of war injuries (intractable scars, neuroma, neuritis, etc.), showing by the presentation of cases that the results achieved were satisfactory. I was pleased to see him recommend strong doses of hard rays, passed through thick filters, because so far as I can see, the future of roentgen therapy lies in strongly filtered rays passing through 4 to 5 mm of aluminum and in massive doses which alone will permit of such a filtration.

TRANSLATIONS & ABSTRACTS

WINTZ, H. A Grounded Roentgen Tube for Deep Treatment. (*Muench. med. Wchnschr.*, Vol. 63, No. 49, Dec. 5, 1916, p. 1719.)

The demands of deep roentgen therapy have more and more crystallized into a demand for high penetration. As a result, the modern roentgen equipment has undergone such vast improvements during the last three years that now electric charges may be sent through the tube with such a plethora of cathode rays that the filtered product constitutes a satisfactory mixture of roentgen rays.

The desideratum of all roentgenologists are homogeneous rays. Homogeneity has become the watchword, especially since it has been possible by measuring devices to establish for each kind of ray a factor of heterogeneity. It is a long way yet to homogeneous roentgen rays, and it is questionable whether they will be produced within a measurable time. The mixture of rays which is furnished by a good modern roentgen tube is a non-homogeneous composite which still widely differs from the gamma rays of radium. Even the Coolidge and Lilienfeld tubes send out strongly non-homogeneous rays.

For this reason we shall have to be content for the present with constructing roentgen instruments and tubes which at least furnish a deeply penetrating, though non-homogeneous mixture of rays, but in abundant quantities. The roentgen tubes with a low vacuum which are now in use, require the presence of a little gas to allow the current to pass through. If the best the tubes are capable of is to be got out of them, the quantity of gas allowed should be just sufficient to let the current push through the tube. If the tube becomes only a shade harder, *i.e.*, if the gas content is reduced, the resistance of the tube is at once increased to such an extent as to compel the spark to travel over the parallel spark gap. If the latter is of sufficient length (38 to 40 cm), proof is furnished of the fact that the tension of the tube is very high and that, consequently, accelerated cathode rays impinge upon the anticathode. Such a tube should always endeavor to use up its gas content and will therefore require constant regeneration which, however, is effected in a mere fraction of a second. (*Muench. med. Wchnschr.*, 1916, No. 11.)

Of course, a performance of this kind can only be expected of well constructed tubes.

There is, however, another point which carries considerable weight in the attainment of the hardest possible roentgen rays, and that is the ionization of the tube. The conception of ionization is as yet but little understood, and for present purposes I may explain it by the fact that the passage of the current and the resultant induction of roentgen rays cause a peculiar arrangement in the gas molecules. There occurs a migration of the disintegrated gas particles which materially facilitates the independent passage of the electric current through the tube. Practically the same refers to the accumulations in the tube and at the glass wall, and these are the very factors favoring the occurrence of gliding sparks along the tube.

Thus, an equipment forcing the induction of highest penetration from hard roentgen tubes means constant danger to the tubes.

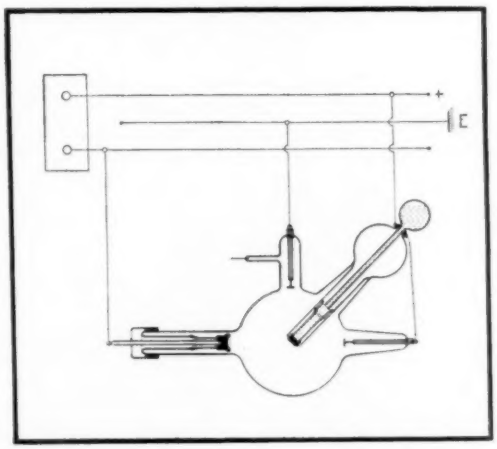

FIG. 1.

Metal parts in the vicinity of the tube may likewise collect an electric charge which may be unloaded on the patient in the shape of very unpleasant electric shocks. Thus, when I determined in our clinic (University Woman's

Clinic, of Erlangen) to confine our treatment to the hardest rays, it became urgently necessary to overcome these drawbacks. The most necessary change was the installation of a non-metallic stand which was supplied by Reiniger, Gebbert and Schall. This, however, helped the tube but little. Ionization and accumulations continued as before. The accumulations at the glass wall and those on the zinc filter a few centimeters away were sometimes so considerable that crackling sparks were exchanged between them, finally perforating the glass wall.

This caused me to experiment with grounding the tube. The localization of the ground conduit offered some difficulties. Without going into particulars, I may say that the direction of the bundle of cathode rays was irritated by the grounding. The best arrangement was finally found to be the following: The starting point of the grounding wire was arranged centrally above the anticathode and allowed to protrude with a plate into the interior of the tube (Fig. 1).

The usefulness of the grounded roentgen tube has brilliantly confirmed our expectations. Up to the present the tube has remained free from electric accumulations, so that it can be touched with the hand when placed in series with the grounded zone. The working of the installation has consequently become very regular, and although such a tube runs with a 38 to 40 cm spark gap, the working is quiet without any unpleasant crepitating and crackling noises and without any danger of sparks being discharged on the patient. This also overcomes almost entirely the danger of tube perforation. A large number of grounded tubes are now installed in our roentgen department and work to our highest satisfaction. I have had this improvement built into the "dura" tube and the "rapid-boiling" tube.

Aside from the incontestable advantages of the grounded tube, there is the important possibility of introducing a specially constructed tube of this kind into the vagina, at least partially. That would enable us to carry out real vaginal roentgen treatments. With the present method of applying roentgen rays through the vagina there are the difficulties of a proper stand and the handling of the speculum, which render it almost impossible to direct the central ray in the desired angle.

EUSTERMAN, GEORGE B. Syphilis of the Stomach: A Clinical and Roentgenographical Study. (*Am. Jour. Med. Sc.*, Vol. CLIII, No. 1, January, 1917, p. 21.)

The author reports 23 cases of syphilis of the stomach which have so far been observed at the Mayo Clinic. He states that it is generally conceded that the clinical picture resembles that of non-specific gastric disease, but the different results of the therapy will dispel the suspicion to such an extent that in cases where atypical gastric disturbances do not respond to ordinary dietetic and medical treatment, the suspicion of syphilis is very frequently justified. Lack of sufficient material has prevented the establishment of a characteristic syndrome, although it is safe to say that the condition has a greater incidence than is commonly supposed.

The diagnosis is usually based on the past history with regard to initial and secondary symptoms, the demonstration of late syphilitic manifestations, and the Wassermann or Noguchi reactions, although the absence of any of these signs does not exclude the disease. The presence of objective signs in other parts of the body would be suggestive, and of the greatest importance is the combination of anacidity, subacidity or achylia with a syndrome approximating but not exactly resembling that of benign ulcer in some respects and that of gastric carcinoma in others. The conclusion that a gastric lesion is necessarily luetic if accompanied by a consistently positive Wassermann reaction is a fallacy, as the coexistence of syphilis with benign and malignant gastric disease has no obvious relationship. There is considerable difference in the gross pathology of benign and gummatous ulcers that is quite apparent to the experienced on ordinary examination. The latter are invariably multiple, have a predilection for the cardia, the lesser curvature and especially the pyloric part of the stomach. Usually, they are associated with perigastric adhesions, proliferative hyperplasia of the gastric walls and other sequelæ resulting in gastric deformity. To the clinician and roentgenologist these characteristics, in conjunction with a markedly altered chemistry, rather suggest carcinoma than benign ulcer callosum.

All of the reported cases showed deformities of variable degree, usually quite marked, with a reduction in size and contour. This was primarily the result of multiple cicatrizing,

gummatous ulcers, or of areas with perigastric adhesions. The degree of involvement was invariably in direct ratio to the duration of infection and gastric disturbance. In 4 of the 10 surgically verified cases there was definite hour-glass deformity. One patient had a stricture at the cardia, at the mid-portion and at the pylorus. In all of the cases, the pars pylorica was the most extensively involved, although in 5 instances the involvement extended up both curvatures to the esophageal opening. In addition, there was marked thickening and stiffness of the walls.

The roentgenograms of two others showed hour-glass contraction, the greater loculus being above. In 3 instances, the ulceration and thickening seemed confined mostly to the posterior wall. In one of the cases, the stomach was described as having a ruffled appearance due to the presence of multiple, cicatrizing, gummatous ulcers.

Gross filling defects, mainly in the lower two-thirds, suggesting extensive disease or involvement, were readily apparent in the other roentgenograms. The one congenital case showed a small, deformed stomach with the normal portion reduced to a sac at the cardia. To the roentgenologist, the combination of gross filling defect, tendency to hour-glass deformity, absence of palpable mass and six-hour barium residue, and the absence of proportionate cachexia, suggests gastric syphilis. Technically, however, such findings can not be differentiated from carcinoma, as there is no characteristic sign which readily enables the clinician to differentiate against some types of serous carcinoma, linitis plastica or sclerotic inflammation secondary to benign ulcer. All things considered, the author holds that the aid of the Wassermann or Noguchi reaction and a roentgen examination are necessary to establish the presence and the specificity of the lesion.

Bissell, Joseph B. Radium in Various Surgical Conditions. (*N. Y. State Jour. Med.*, Vol. XVI, No. 12, December, 1916, p. 591.)

In this article Dr. Bissell describes the effect of radium on widely different growths, from corns to fibroids, showing that the almost miraculous effect of this agent is not confined to the superficial tissues. He selects nine cases on account of the interesting variety of the affection. In the first case, an inflamed thick, hard corn on the little toe entirely disappeared, without causing local irritation to the neighboring skin, after six applications of unscreened radium, each of twenty minutes' duration, four or five days apart. An inflamed corn on the ball of the foot which had been repeatedly cauterized and cut out, without permanent effect, was treated with 10 mg of radium for fifteen minutes. Five days later a callous mass was removed in the centre of the corn, and into the hole thus made a tube containing 25 mg of radium in a silver case was applied for fifteen minutes, the surrounding skin being protected by 1 mm lead and 2 mm rubber. When this procedure had been repeated several times at six days' intervals, the patient was entirely relieved.

A fibroid of the uterus became so small after application of 50 mg of radium in the cervix for five hours and of 15 mg for ten hours five weeks later, that at the end of three months an operation was no longer found necessary. The symptoms of hemorrhage, pain and pelvic discomfort had likewise disappeared. A more checkered career had the healing process in a 47-year old woman with several fibroids. Here the application of 50 mg of radium in one tube for twenty hours caused considerable nausea, headache, sore throat, cough, and a temperature as high as 104°. When this condition had subsided after a week, the cervix was found congested, tender, swollen, easily bleeding. Besides, there was a profuse discharge. Application of 50 mg for ten hours again caused nausea but no particular pain. Two days later the temperature had risen to 105.6° and there were severe headache, rectal tenesmus, slight diarrhea, frequent and painful urination and other symptoms of local irritation. After a stormy time, lasting three weeks, only a vestige of the anterior fibroid remained, and the os was soft and patulous with no evidences of fibroids. A radium burn of the uterus had caused a severe infection ascending one or both ureters, and a moderate proctitis. Eventually the patient made a complete recovery.

A one-inch keloid on the right breast of a young woman, following a scald, was treated for thirty minutes with 15 mg of radium in varnish applicator, causing a slight blister and swelling. A week later, varnish applicator and tube of 10 mg with rubber screening were applied for ten minutes; five weeks later, when

the tumor was found to be reduced to half its former size and very much thinner, two 5 mm varnish applicators were applied for twenty minutes. When the patient reported three months later for final examination, there was only a white scar remaining.

A 17-year old girl had a keloid scar, four inches long and at points one-quarter of an inch high, running across the left cheek. Radium was applied for twenty to thirty minutes once a week fifteen times, when the patient abandoned treatment. The keloid had softened and only the soft scar remained.

A 37-year old woman had a toe amputated for osteomyelitis, the wound refusing to heal because of dead bone. There was an indolent sinus with protruding flabby granulations. Two ten-minute applications of 10 mg radium each resulted in a cure within less than a month.

Septic infection of the knee-joint in an old man, involvement of the lower end of the femur, patella and tibia, with destruction of cartilage, with several sinuses and copious seropurulent discharge, gave rise to such a serious condition that amputation was advised which patient refused. Radium effected a cure with an ankylosed but useful knee-joint. The treatment consisted in (1) 100 mg radium in five tubes applied in the different sinuses for ten minutes; (2) twelve days later: radium applied as before; (3) six weeks later: another application of radium for twenty minutes and the remainder of 100 mg radium strapped around the joint at equidistant points; (4) four weeks later, a small pocket of pus below the patella was incised and evacuated, which healed in about two weeks. There was progressive improvement during the various applications.

A similar history is presented in the following case. A laborer, 43 years of age, had his knee caught between a car and a packing box, resulting in purulent sinovitis and such a bad condition that amputation was advised. Patient refused amputation and radium effected a cure within about four months with ankylosis at about an angle of 20 degrees. One hundred milligrams of radium in divided doses were applied through the tube in the knee-joint for twenty minutes. Three more applications were made at appropriate intervals as healing progressed.

The last case described occurred in a seven-year old girl with a swelling over the first metatarsal bone. Roentgen examination showed an osteitis in the first and second metatarsal bones which two months of surgical treatment failed to improve. Three radium applications, lasting ten minutes each, resulted in gradual improvement and complete cure in seven weeks. The case was apparently one of tubercular sinovitis.

LOEHNBERG, ERNEST, AND DUNKER, FRITZ. Two Cases of Very Grave Congenital Defects of All Extremities. (Tenth Anniversary of the Cologne Medical Academy. Ref. *Zentralbl. für Roentgenstr.*, 1916, Nos. 5 and 6, p. 155.)

The first case concerns a newly born child; the second a man 61 years of age. In both cases the upper arms and femora are only preserved to the upper thirds. Elbow and kneejoints, lower arms and legs, hands and feet are missing. By way of contrast there is an apparently undisturbed development of the head, neck and trunk. The roentgenograms show that in the second case the humeri are distorted in varus form and extremely short, consisting almost of nothing but head, neck and tuberculum majus and being elongated after the form of stalactites in the rudimentary shaft. In the first case the upper part of the humeri and shafts are well developed, while the lower epiphyseal part and the condyles appear more or less hypoplastic. The general symmetry is rudely interrupted by the total absence of the right femur, and in view of the fairly good development of the soft parts of the femoral stump this must be regarded as an important guiding point in determining the origin of the deformity. The authors look for the cause in abnormal uterine pressure contributing to the grave mutilation of all extremities without materially interfering with the normal delineation of the cleft line.

BOOK REVIEWS

LOCALISATION ET EXTRACTION DES PROJECTILES. By L. Ombrédanne and R. Ledoux-Lebard. Masson & Cie., Paris, 1917.

In spite of the excessive demands made upon the services of roentgenologists in war practice, the authors have found it possible to compile a thoroughly instructive book of 349 closely printed pages with 225 text illustrations and 8 roentgen plates, setting forth the various methods devised and extant for the localization and extraction of foreign bodies and their value in actual practice under the varying conditions of the injuries sustained. The work has been adequately divided between two authors equally eminent in their respective fields of endeavor, Ombrédanne taking charge of the surgical aspect of the subject, while Ledoux-Lebard needs no special introduction to our readers as one of the most distinguished roentgenologists of France. By far the larger part of the book is devoted to the roentgen rays, although the cases where foreign bodies can be properly dealt with without their aid have not been overlooked.

The book is divided into three parts, the first dealing with the physical properties of the roentgen rays and the mechanical requirements for their most advantageous application. In the second part, the details of the various methods, their indications, advantages and disadvantages are carefully described with a competent and discriminating mind, and while it seems but natural that the authors should be especially familiar with the practical application of the methods devised and favored in their own country, they have not overlooked the claims of other countries, notably of America, where they single out the names of Lester Leonard, Case and Pfahler as worthy of special mention, more especially in connection with the utilization of the stereoscope in the plastic visualization of foreign bodies.

The third part deals with the extraction of foreign bodies, a subject which is naturally subdivided into methods with and without the use of the roentgen rays, the latter acting as a screen guide to the surgeon.

In view of the recent rapid multiplication of methods for the localization of foreign bodies in the human anatomy, due not only to the technical advance of roentgenological knowledge but also to the occurrence of countless cases of war injuries which make the requisition of some such method a matter of daily and indeed hourly occurrence, there is no gainsaying the fact that, if ever a book was published at the time it was needed, that book is the one under review.

THE JOURNAL OF UROLOGY

We have received the announcement of a new publication entitled *The Journal of Urology* which has been founded with the intention that it shall become the archives in the United States for papers dealing with the urinary tract and correlated subjects. While America has been foremost in the development of urological knowledge, we have had no special journal in which these advances have been recorded. The situation has resulted in many important experimental papers, which have recently appeared, being widely scattered in various publications. It is the purpose of the new journal to centralize the publication of articles on urology. Interesting papers of a historical nature will also be presented. The first issue appeared in February, 1917, under the editorial supervision of Dr. Hugh H. Young, of Baltimore, and a competent executive committee.

LA RADIOLOGIA MEDICA

La Radiologia Medica, a monthly publication, is the official organ of the Italian Society of Medical Radiology, edited by Dr. Prof. Felice Perussia and a large editorial staff. The current issue, Vol. III, Nos. 3 and 4, is a good sample of this very commendable publication. It contains the following articles: "Partial Hepatoptosis," by Perussia; "Radiologic Study of the Influence of Alcohol on Gastric Motility," by Bosio; "Two Cases of Complete Fracture of the External Tuberosity of the Tibia," by Taparelli; "Physics of the *X*-ray," by Ceresole; "Radiologic Service in the War (a type of *x*-ray automobile)," by Boidi-Trotti; "Obligatory Attendance in a Course of Radiology in the University," by Prof. Sgobbo. There are also sections on new books, proceedings of the Italian Society of Radiology.

In size of page and general appearance this journal resembles the *Fortschritte*.

A review of the article on alcohol will appear in our abstract columns.